The Journal of Thoracic Surgery

VOL. 35 JUNE, 1958 No. 6

Original Communications

AN EVALUATION OF IMPLANTATION OF THE INTERNAL MAMMARY ARTERY INTO THE MYOCARDIUM OF PIGS AND DOGS

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SURGICAL efforts to introduce a new blood supply into the myocardium have been numerous and varied. The internal mammary artery implant as first described by Vineberg[15] in 1946 seemed a simple but intriguing approach to the problem. The reported observation that an open vessel implanted into myocardium remained open appeared to be a biologic phenomenon of great importance and deserving of intensive investigation. Consequently, when we became interested in the problem of myocardial revascularization, we were particularly interested in the procedure of internal mammary artery implant. Knowing the difficulty of evaluating any of these procedures in the human being, we began our investigation in the laboratory animal, using the weanling pig as our experimental animal.

The pig was selected primarily because its coronary circulation is of the end-artery type and very closely approximates that of the human being.[3] It has been shown that ligation of the left anterior descending (LAD) artery in the pig results in almost 100 per cent mortality within 15 minutes.[3] Logically, a highly consistent test ligation of the LAD artery might prove to be a valuable yardstick for determining the efficacy of any operative procedure in improving the coronary blood supply.

A total of 25 pigs survived the implantation of the left internal mammary artery into the myocardium of the left ventricle. Eleven of these were operated upon after a 6- to 12-week period and a test ligation of the LAD artery was

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Received for publication Sept. 20, 1957.

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performed. Of this group, 10 died promptly of ventricular fibrillation, suggesting a failure of the implant to protect the myocardium from the effects of LAD artery ligation. Post-mortem examination in each of these 10 animals revealed gross and microscopic occlusion of the implanted internal mammary artery. Fourteen of the pigs with internal mammary implants grew too large to handle for secondary operation in our laboratory. These were slaughtered at an abattoir and the hearts removed for examination. In no instance was an anastomosis demonstrable between the internal mammary implant and the coronary circulation. Only one pig in the series of 25 (4 per cent) could be considered to have a successful internal mammary implant. This animal survived the test procedure of LAD ligation and gross anastomosis between the patent internal mammary and branches of the LAD artery was demonstrable by injection of colored vinyl plastic.

It was apparent that this series was inconclusive. In using a different experimental animal, the conditions of Vineberg's experiments were not reproduced. The pigs were all young animals and this might properly be considered an unfavorable feature in the test of the procedure. The weanling pigs responded to open chest surgery with an extensive dense fibroblastic reaction. Some of the adhesions at the operative site were almost cartilaginous in consistency. This was equally true when a midsternal extrapleural approach was perfected. Another serious objection to the validity of the results was the failure to produce myocardial need. In addition, it was noted that Vineberg's results improved as he and his associates gained technical proficiency. It seemed reasonable that we, too, might expect a higher number of "takes" as our familiarity with the procedure grew. Accordingly, a new series was begun in dogs, duplicating the conditions of Vineberg's experiments in every detail.[15]

PROCEDURE

Healthy mongrel dogs weighing 25 to 45 pounds were operated upon through the left chest under intravenous Nembutal anesthesia. Pulmonary ventilation was maintained by positive pressure oxygen administered by a modified Emerson* resuscitator through an intratracheal tube. Myocardial need was created by partially ligating the LAD branch of the left coronary artery over a No. 23-gauge needle (Fig. 1). The left internal mammary artery was carefully dissected free between the fourth and sixth interspaces. The distal end was divided and ligated preserving a suitable intercostal branch. The myocardial tunnel in the left ventricle was made with a small curved clamp and planned to prevent the possibility of angulation of the internal mammary as it was pulled through. Just prior to burying the implant, the selected intercostal branch was divided flush with the artery so that there was free and vigorous bleeding into the tunnel. The distal end of the implant was then anchored to the exit of the tunnel by a suture-ligature. The pericardial fat pad was utilized to prevent adherence of the internal mammary to the denuded sternum rather than as an additional source of blood to the myocardial surface.

*J. H. Emerson Company, Cambridge, Mass.

Animals surviving the procedure were sacrificed 2 to 3 months later. Flow studies were performed through the isolated implant in all animals not having obvious gross occlusion of the implanted vessel. Normal saline solution at 120 mm. mercury pressure was used in the flow studies. Whenever an apparent flow through the implant was obtained, the saline was colored with methylene blue to locate leaks and to look for myocardial staining. Schlesinger's[13] mass was injected into every implant with a demonstrable flow. All such specimens were sectioned and studied microscopically along with representative specimens of grossly occluded implants with no flow.*

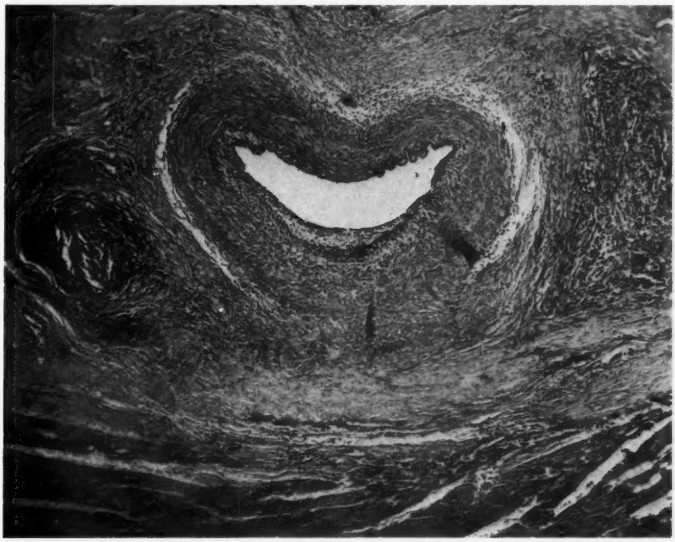

Fig. 1.—*Dog No. 116.* Photomicrograph of the LAD artery distal to a partially occluding ligature producing myocardial need. The lumen is still patent, although narrowed. There is minimal intimal thickening.

RESULTS

Nineteen of 23 dogs survived the operative procedure over the experimental period. All 4 mortalities occurred within 2 weeks of operation and were the result of the partial occlusion of the LAD artery. One implant of the remaining 19 was demonstrably patent by all criteria (Dog. No. 116). There was a flow of 6.5 c.c. saline per minute at 120 mm. mercury pressure in the living animal. The vessel was grossly and microscopically patent. Schlesinger's mass injected into the implant was seen to enter the LAD artery and its branches (Fig. 2). In this one animal, a significant anastomosis developed between the implanted internal mammary artery and the left coronary artery system. The other 18 implants were occluded, giving a 5.3 per cent rate of successful internal mammary artery implants.

*We gratefully acknowledge the assistance of Drs. Tobias Weinberg, Robert Solomon, and Alma Kelly of the Department of Pathology in the preparation and interpretation of this material.

Fig. 2.

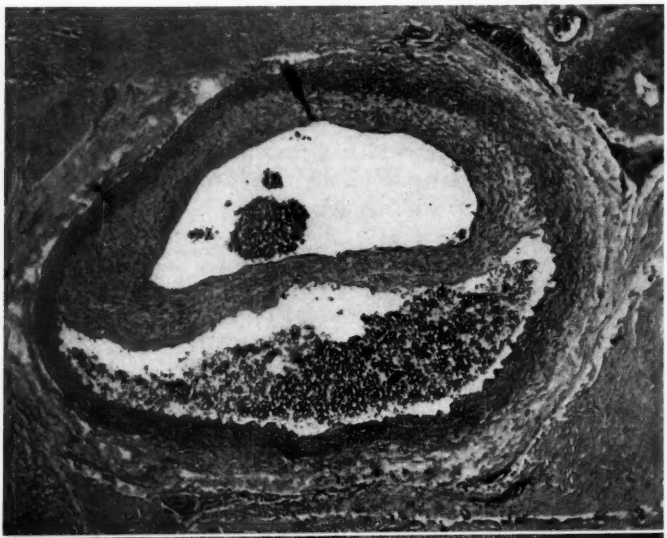

Fig. 3.

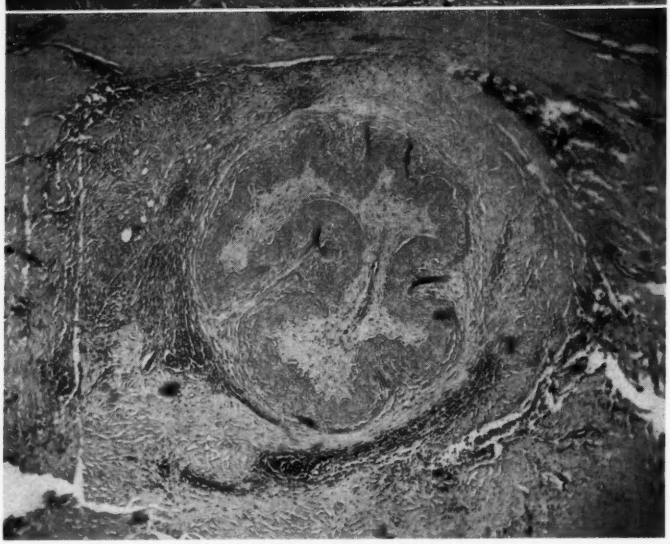

Fig. 2.—*Dog No. 116.* Section through the myocardial tunnel demonstrating a widely patent implanted internal mammary artery. There is slight intimal thickening with dissection of the intima from the media. Whether or not this is an artefact is not known. Note the Schlesinger's mass present in both the true lumen and false passage as well as within the small branches nearby.

Fig. 3.—*Dog No. 143.* Section through the internal mammary artery within the myocardial tunnel. In spite of an apparently successful flow study, the lumen is completely obliterated. The obliteration is due to compression of the vessel wall as well as intimal proliferation.

Fig. 4.

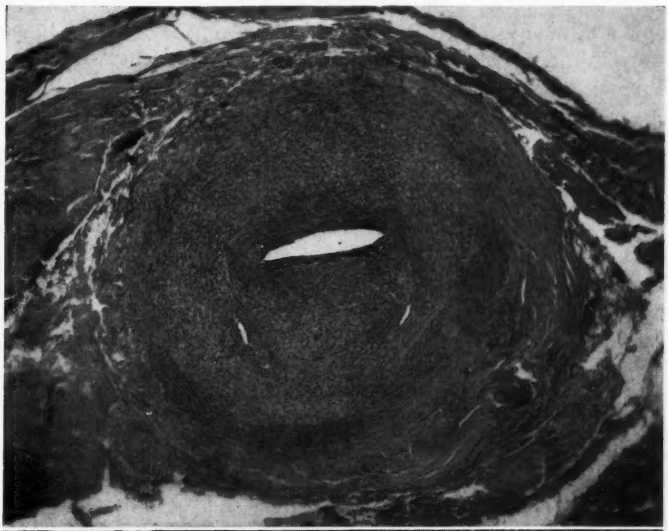

Fig. 5.

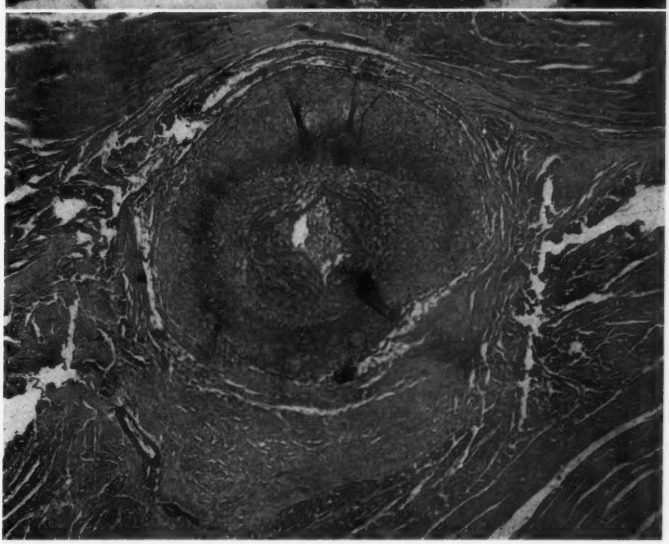

Fig. 4.—*Dog No. 208.* Section through the internal mammary artery proximal to the myocardial tunnel. Note the extensive intimal proliferation resulting in marked narrowing of the lumen.

Fig. 5.—*Dog No. 167.* Section through the internal mammary artery within the myocardial tunnel. Note the extensive intimal proliferation and marked narrowing of the lumen. There is Schlesinger's mass present within the lumen of the implant but none demonstrable in the small branches nearby.

One animal (Dog No. 143), in which a 3.0 c.c. per minute flow was obtained, proved to have an occluded implant on subsequent Schlesinger's injection and microscopic study (Fig. 3). This apparent discrepancy proved to be due to minute leaks in the internal mammary artery prior to its entrance into the myocardial tunnel. The addition of methylene blue to the perfusate eliminated this source of error in subsequent experiments.

DISCUSSION

It is difficult to evaluate objectively the efficacy of a revascularization procedure on the human myocardium. Obvious clinical improvement may follow an operative procedure but may be entirely unrelated to the specific operation employed. The precise evaluation of an advocated operative procedure by suitable laboratory study is of primary importance.

In our hands, the implantation of the internal mammary artery into the myocardium was not a satisfactory method for improving the blood supply to the heart. An occasional successful result was obtained, but not with sufficient frequency to warrant our employment of this procedure in patients. Although other investigators have reported similar findings, their evidence was inconclusive. Glenn and Beal[6] reported results in 6 dogs observed for a period of 1 to 6 weeks. Their operative procedure differed considerably from that advocated by Vineberg and their adverse findings must be regarded as inconclusive. Bakst and co-workers[1] reported poor results with implantation of the internal mammary artery but used only 9 animals in none of which was myocardial need established. In our study, the usual criticisms were obviated. The size of the series was adequate. The technique employed was exactly as described by Vineberg. Myocardial need was established. The operating surgeons had previous experience in internal mammary implants and were not attempting a new procedure for the first time.

The often-described phenomenon of intimal proliferation was consistently present (Figs. 4 and 5) and could be found to some degree even in the successful implant. This may eventually be circumvented by the use of a larger vessel,[5, 10, 12] or an artificial graft.[14]

In performing a flow study on an implanted vessel, it is essential to ascertain exactly where the perfusate is going. An anastomosis via adhesions to the sternum or mediastinal fat may result in a misleading flow study. Small leaks from such vessels which have been divided are readily detected when the perfusate is colored with methylene blue. Failure of the dye to discolor the myocardium is additional evidence that flow through the implant is not synonymous with flow into the heart muscle.

Injection of the implant with Schlesinger's mass is important in establishing the caliber of the anastomotic channels. The mass will not traverse a vessel with a diameter less than 40 microns.[13] Only Dog No. 116 was successfully injected with this material.

Although these first attempts to introduce a new blood supply into the insufficient myocardium have been disappointing, investigation of the value of certain modifications of the original procedure are in progress in this laboratory.

SUMMARY

1. Implantation of the internal mammary artery into the myocardium was not a satisfactory revascularization procedure in our hands.

2. A significant anastomosis was demonstrable in only one of 19 dogs (5.3 per cent) subjected to implantation of the internal mammary artery after partial ligation of the left anterior descending artery.

3. Successful anastomosis was demonstrable in only one of 25 pigs (4 per cent) subjected to a similar procedure.

(*For References, see page 717*)

MAINTENANCE OF PATENCY OF CAROTID ARTERIES IMPLANTED INTO THE MYOCARDIUM

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IN 1946, Vineberg[15] introduced the ingenious procedure of placing the left internal mammary artery into a myocardial tunnel. His subsequent reported experiments presented evidence to indicate that such an implanted vessel remained patent and developed arterial connections with the coronary vascular system, thus furnishing an extracardiac source of blood to the myocardium.[16, 17] Not only was there an anatomic connection but, also, this extracardiac arterial blood was capable of protecting the heart muscle against both the mortal and morbid effects of coronary artery occlusion in experimental animals and in man.

This procedure has not attained widespread acceptance for, in the hands of other investigators, the results were not the same as his. Glenn and associates[7] observed that the new vessels which formed were small, resembling the capillaries seen in granulation tissue, and tended to disappear some time after the operation. Bakst[1] and Maniglia[9] and their co-workers implanted the mammary artery into the myocardium of 9 dogs and, at the time of sacrifice 6 months later, found that the lumen was obliterated to 10 per cent or less of its original size in 7 of the 9 animals, while in the remaining 2 dogs it was 30 per cent and 50 per cent patent. The physiologic studies from the same laboratory gave no indication of any benefit from the implantations. Beck[2] has also evaluated this procedure in dogs, and has been unable to find any evidence that a significant amount of extracardiac blood is delivered by the implanted vessel. On the other hand, Sabiston, Fauteux, and Blalock,[12] using a larger artery (carotid) and a somewhat different technique, were able to demonstrate that the lumens of the implanted vessels remained patent in 28 of 32 dogs. However, when they measured the amount of blood supplied to the heart muscle by the implanted vessel, the values averaged no more than 5 c.c. per minute.

Because of the contradictory nature of the reports, and because the concept of arterial implantation into the myocardium appeared to be a biologic phenomenon, sufficiently unique and important to merit careful investigation, we began to evaluate the procedure in this laboratory 3 years ago. The early

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This work was supported by research grants (H-2032 and CY-2478) from the National Institutes of Health, Department of Health, Education and Welfare and from the Landsberg Memorial Fund, Sinai Hospital of Baltimore, Inc.

Received for publication Oct. 25, 1957.

results revealed that in 25 young pigs and in 19 dogs, the lumen of the implanted internal mammary artery became obliterated within 4 weeks in all but one animal of each species.[11] The present paper presents the experiments which were carried out in an effort to understand the cause of the intimal proliferation and the consequent obliteration of the artery which had been implanted into the myocardium. It will be shown that when continuous flow through the implanted artery is maintained by plunging the open bleeding end of the vessel into the lumen of the right ventricular cavity, the implanted vessels do not develop intimal proliferation.

EXPERIMENTAL STUDIES

Control Group.—The use of an artery larger than the internal mammary was suggested by the notion that if the lumen were narrowed to 50 per cent or even 75 per cent of its original size, an adequate flow of blood still might be supplied to the heart muscle. This thought was also present in the minds of other investigators, who have since presented the results of their work.[5, 12] Consequently, the left carotid artery was mobilized through a vertical cervical incision, 2 cm. to the left of the midline. The left chest was then entered through the fourth intercostal space and, with blunt dissection from within the thorax and the neck, the transected carotid artery was freed sufficiently to bring it into the pleural cavity. The pericardium was incised and after one tangential opening, 4 mm. in diameter, was made in the carotid artery, the vessel was pulled into a tunnel in the left ventricular myocardium. An inspection was made to be certain that the artery was not significantly kinked at its origin as it was directed downward toward the heart, and that it was not obstructed at the point of entry into the myocardium. The pericardium was loosely reapproximated and the chest wall closed.

This operation was performed on 14 dogs which served as the control group. At the end of one month the animals were sacrificed, and the hearts with their implanted vessels were studied microscopically. In 13 of the 14 dogs, the lumen of the implanted vessel was reduced to 25 per cent or less of its original area. This was caused by intimal proliferation in 11 of the animals, while in the 2 others a thrombus was responsible for the occlusion. In one dog of this group, the portion of the vessel within the myocardium had retained 80 per cent of its original lumen, but the more proximal portion was narrowed to 20 per cent and was the site of a fresh thrombus. Thus, at the end of one month, these implanted carotid arteries were unable to deliver a significant extracardiac source of blood to the myocardium.

Implantation Modified to Include Placing Carotid in Right Ventricle.—The high incidence of intimal proliferation and thromboses as reported by others,[1, 2, 7, 9, 11] who used the internal mammary artery, was confirmed by our experiments in which a larger vessel was implanted by the standard operative technique. Our attention was directed, therefore, to the problem of trying to understand the cause of the intimal proliferation. Previous work had suggested that the maintenance of flow through the implanted vessel was important.[4]

This was shown by the procedure of dividing the internal mammary artery proximally after it had been freed from the chest wall, carrying the artery through a ventricular tunnel, and anastomosing the proximal end of the vessel to the left atrium. By such a technique Carey and associates[4] were able to demonstrate a marked reduction in the occurrence of intimal proliferation as compared with the control series. The procedure which we instituted was to perform the same operation as described in the control group above, but to place the distal end of the carotid artery with its open end into the right ventricular cavity. This procedure is illustrated in Fig. 1.

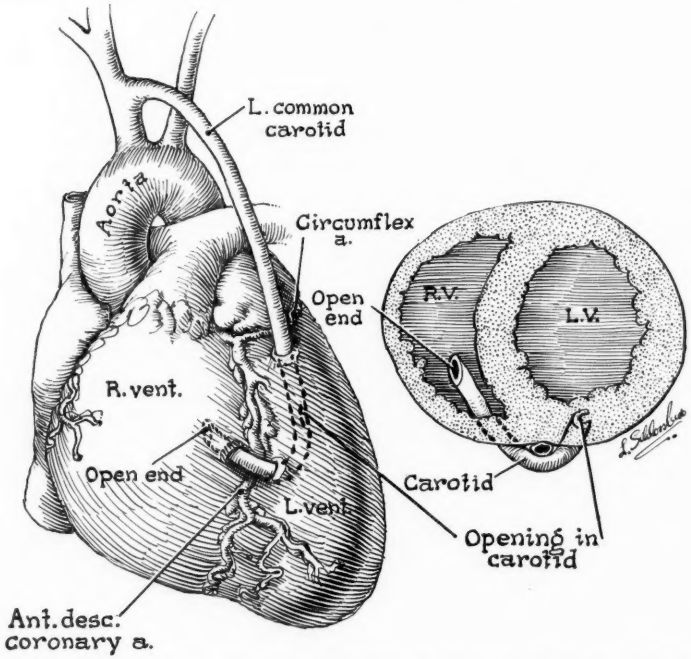


Fig. 1.—Illustration of the double implant type of operation where the carotid artery with one tangential opening is implanted into the left ventricular myocardium and the distal open end of the vessel is placed in the right ventricle.

Effect of Cardiac Contraction on the Implanted Vessel.—It seemed advisable to determine whether the trauma which the artery sustained by repeated ventricular contraction was responsible for the intimal proliferation. Therefore, the procedure just described was carried out on 4 dogs with the exception that no tangential opening was made in the artery as it passed through the myocardial tunnel of the left ventricle. Thus, we could determine the effect of the cardiac trauma alone. In 3 of these 4 animals, which were sacrificed at the end of one month, the artery appeared grossly as soft and patent as the day it had been implanted. This was in striking contrast to our control group, in which the arteries were thickened, rigid, and almost completely obliterated.

The fourth dog was a technical failure in that the distal end of the implanted vessel had retracted into the right ventricular myocardium so that flow into the right ventricular cavity was not possible. The lumen of this implanted vessel was occluded throughout its course in the myocardial tunnel of the left ventricle resembling the arteries in the control group. These experiments appeared to support the argument that the trauma to which the implanted artery was subjected by the repeated muscular contractions did not result in intimal proliferation, as long as blood was flowing through the vessel.

Findings of Implantation With Flow Into Right Ventricle.—In 29 dogs the carotid artery was implanted into the left ventricular myocardial tunnel with a tangential opening having been made in the artery, and the distal open end of the artery was placed in the right ventricular cavity. Twenty-two of the animals were sacrificed at 1 to 3 months postoperatively, while 7 were followed for 6 to 10 months. A period of 3 to 4 weeks was considered the minimal lapse of time for evaluating the fate of the implanted arteries since, in the control group, the lumens were always obliterated by that time interval. Unexpected obstruction was noted in the distal end of the artery in 14 dogs. In 6 instances, the vessel was not placed accurately because of faulty technique, and it was found to be occluded because it had retracted into the ventricular muscle or was blocked by the papillary muscle. Occasionally (4 dogs) a satisfactory preparation showed a large polypoid thrombus which occluded the end of the artery. Such an obstruction to the distal end of the implant lying in the right ventricular cavity is shown in Fig. 2. In the other 4 dogs, the end of the artery was occluded without an obvious cause. These 14 dogs, therefore, did not have blood flowing through the implanted artery into the right ventricular cavity, and thus represented a preparation similar to the control group. They may be considered as "accidental controls." In the remaining 15 animals, the lumen of the vessel in the right ventricular cavity was either entirely open or had become narrowed during the postoperative period, but had apparently permitted continuous flow of blood through the vessel. These represent the "test animals" which can be compared with both the control and accidental control groups. The desired result of an open distal end which permits blood flow into the right ventricular cavity is shown in Fig. 3.

Results in the Accidental Control Group.—The 14 dogs in this group revealed that the lumen of that portion of the vessel passing through the left ventricular myocardium was reduced to 25 per cent or less of its original size in 11 animals, while in 3 it was 75 to 100 per cent patent. This suggests that perhaps the distal end of the vessel was delivering blood into the right ventricular cavity for some period of time during the postoperative course of the latter 3 animals. However, when these findings are combined with the control group of 14 dogs, there were only 3 dogs among the 28 (11 per cent) in whom the lumen of the implanted arteries remained patent one month after operation. These results were obtained in all those animals with an implanted carotid artery, which had one good-sized tangential opening in the implanted portion of the vessel and which were occluded distal to the tunnel.

Results in the Test Animals With Flow Into the Right Ventricle.—These 15 dogs were considered satisfactory for evaluating the effect of continual blood flow through an implanted vessel. In contrast to the findings just recorded, all of these vessels were soft and pulsatile. On microscopic examination, the lumens were found to be from 80 to 100 per cent patent. Table I summarizes

Fig. 2.

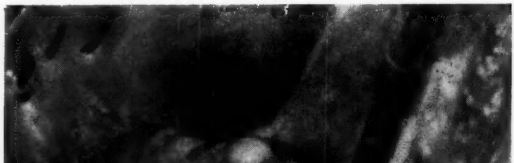


Fig. 3.

Fig. 2.—Photograph showing an obstruction by a thrombus of the distal end of the implanted vessel lying in the right ventricular cavity. The external portion of the carotid is shown on the right, lying on the surface of the left ventricle. It passes through the cut myocardium of the right ventricle and is shown lying in the cavity. The attached thrombus is seen on the left. When this prevents continuous flow through the carotid artery, the implanted vessel shows marked intimal proliferation.

Fig. 3.—In this specimen, the desired result of an open distal end of artery is seen. The artery is lying in the center of the field within the right ventricular cavity just below the pulmonic valve. The implanted portion remains soft and patent, with little or no intimal proliferation.

the results in control and test animals. When the 3 dogs in which the effect of muscular contraction on the artery are included with this test group, it may be noted that 100 per cent of the 18 implanted arteries remained patent when they had an open distal end to allow continuous flow.

That anastomotic connections with the coronary circulation did develop in some of these animals is shown in Fig. 4. When Schlesinger's mass was injected

TABLE I. PRESERVATIONS OF PATENCY IN THREE TYPES OF CAROTID IMPLANTATIONS

GROUPS*	TOTAL NO. OF DOGS	0-25% PATENCY	80-100% PATENCY
		NO. OF DOGS	NO. OF DOGS
1. Controls	14	14	0
2. Accidental controls	14	11	3
3. Test	15	0	15

*The control group represent the animals which had a standard type of implantation of the carotid artery into the myocardium of the left ventricle. The accidental controls were those animals in which the distal open end of the artery was placed in the right ventricle, but which was obstructed due to thrombosis or faulty technique. The test group are those dogs with a persistent open end of the implanted vessel delivering blood into the right ventricle.

into the implanted artery, the entire left coronary arterial system was seen to fill with the blue dye. This animal was sacrificed 9 months after implantation. In 11 of the 15 test preparations either a flow study or a Schlesinger's mass injection, or both, were done. The 6 flow studies performed did not reveal any significant amount of blood flow. Six of the 8 injected specimens had good anastomotic connections between the implanted vessel and the coronary arterial tree. However, flow studies done in 3 of these 6 were disappointing in that they only demonstrated 1, 3.2, and 9 c.c. per minute.

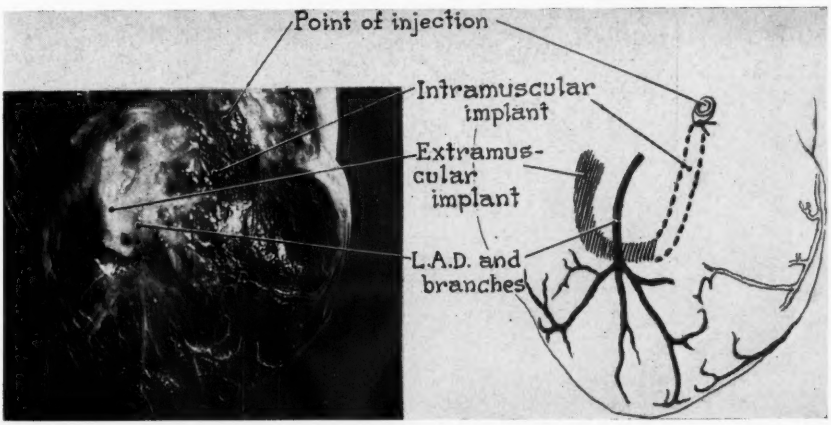
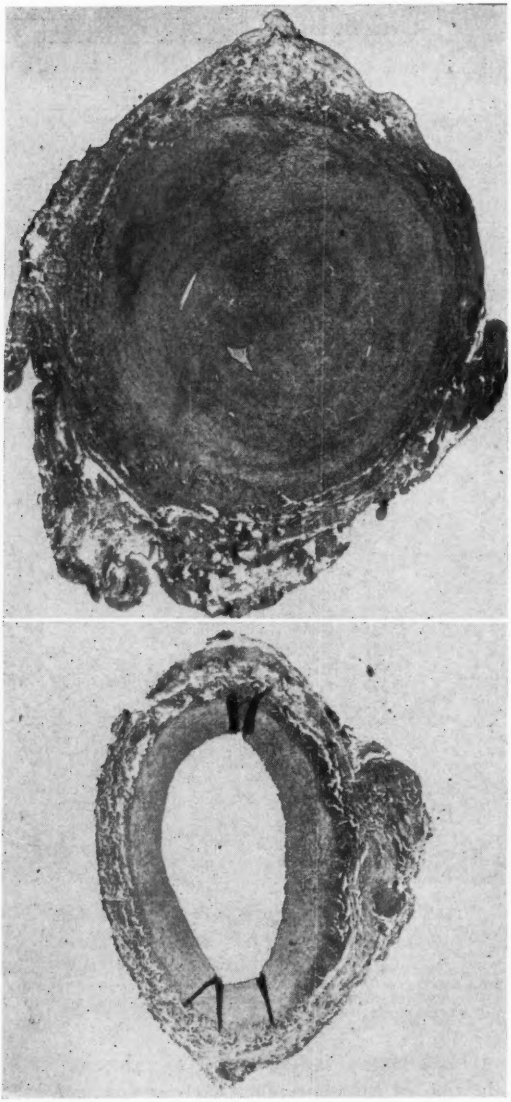


Fig. 4.—Bas-relief photograph. Schlesinger's mass has been injected into the implanted carotid artery 9 months after its placement. The blue-stained mass filled many of the branches of the anterior descending branch of the left coronary artery. On the right is an interpretive diagram, which helps to overcome the deficiencies of the black and white print. The heavy markings indicate those vessels which were seen to contain the blue mass. Similar findings were noted in 6 of the 8 dogs in which such injection studies were done. However, the presence of these connections between the implanted and the coronary arteries was not associated with a significant amount of measured blood flow.

Details of the Pathologic Findings.—An examination was made of each heart at the termination of the experiment. Gross inspections were made and blocks were taken for microscopic examination from the carotid artery proximal to the implant, from the artery within the tunnel, at a point distal to the tunnel and when the double implant was made, adjacent to the site of egress into the right ventricle. Sections were stained with hematoxylin and eosin and with a Weigert elastic-van Gieson stain where indicated.

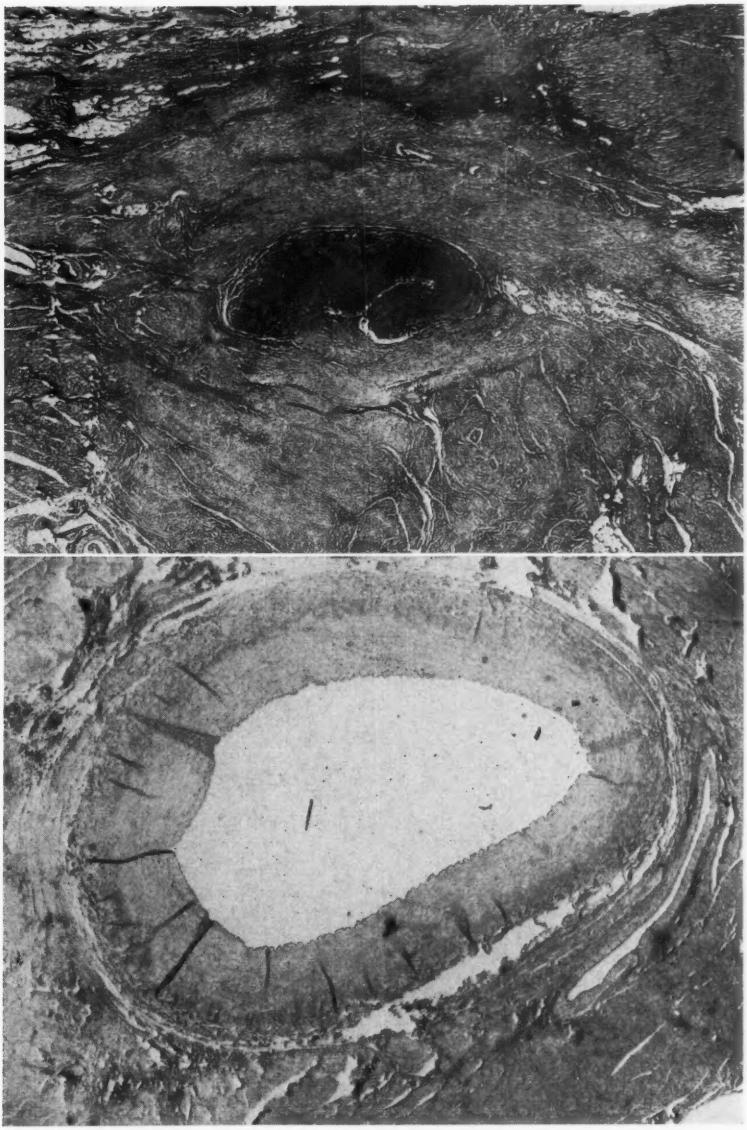
A.



B.

Fig. 5.—*A*, Control preparation; artery proximal to the tunnel, 7 weeks postoperative. The vessel is obliterated by intimal proliferation and organized thrombus. *B*, Test preparation with carotid terminating in right ventricle; artery proximal to myocardial tunnel, 7 months postoperative.

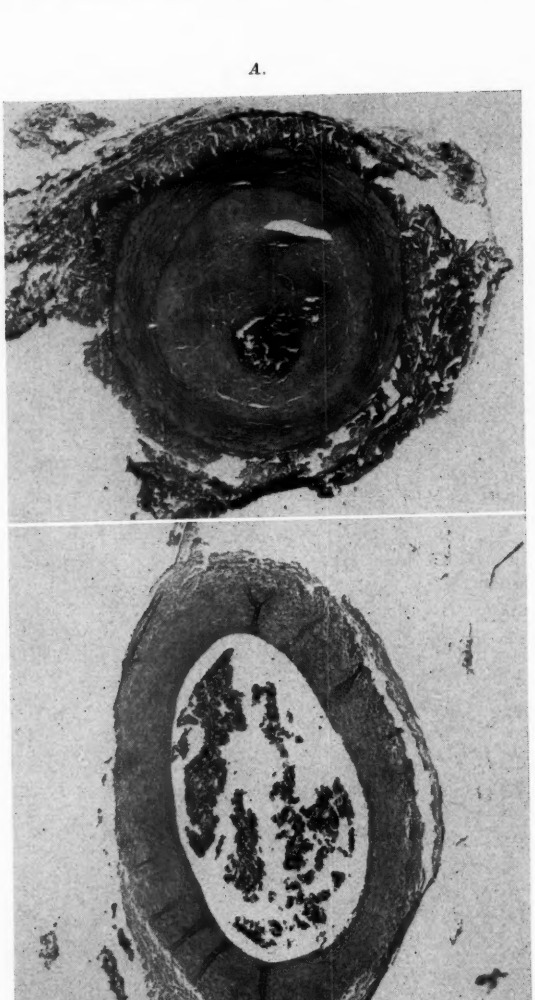
A.



B.

Fig. 6.—*A*, Obliterated carotid artery in a control animal with the artery lying within the intramural tunnel, 7 weeks postoperative. *B*, In contrast, the artery in the test animal with free flow into the right ventricle shows minimal intimal proliferation, 7 months postoperative. The perivascular scar formation is notably small.

A.



B.

Fig. 7.—Artery distal to tunnel in control animal showing obliteration of the lumen and organization of the thrombus after 7 weeks. *B*, Wide open vessel distal to tunnel, proximal to right ventricular implantation; usual experimental result after 7 months.

As others have observed, no hematomas were noted about the vessels in the myocardium even at the points where openings had been produced. There was insignificant phagocytized hemosiderin, but an occasional heart exhibited calcifications adjacent to the implant. The arteries in the tunnels were surrounded by collagenous scar tissue but this reaction, present also in the controls, was variable and did not depend on the duration of the experiment. In some instances there was surprisingly little scar tissue formed about the vessels.

Obliteration of the implants occurred in the controls and "accidental controls" by several mechanisms. Intimal proliferation was present to some extent in all instances, featured by a growth of loose-textured, weblike connective tissue. This was accompanied by some splitting of the internal elastic lamella. Thrombosis occurred in one half of the cases with early or late organization depending upon the duration of the experiment. The thrombotic masses formed on the intimal elevations and also on areas of necrotic intima for, in several hearts, the walls of the carotids were necrotic from adventitia to intima. It seems highly unlikely that the recanalizations present in some of the preparations of the control group, which give the appearance of having a small lumen, could carry any significant amount of blood into the heart.

A striking contrast was noted between the carotids of the test animals where flow of blood into the right ventricle was effected and those ending blindly. This is shown in the illustrations. Fig. 5 is a comparison of the arteries proximal to the myocardial tunnel in a typical experiment. Intimal proliferation is insignificant in the test animal. The arteries within the tunnel and distal to it are compared in Figs. 6 and 7. Furthermore, in some of the test preparations there were strikingly prominent vessels about the implant as long as 6 months postoperatively, the presence and size of which could not be assigned to granulation tissue. These vessels which had been injected through the carotid implant were connected with the coronary circulation (Fig. 4). Further investigation of the factors involved in this encouraging finding are now in progress.

DISCUSSION

The discrepancies which have arisen in the several laboratories which investigated the procedure of arterial implantation into the myocardium are difficult to explain. Vineberg[17] has emphasized that two principles are of utmost importance in the maintenance of patency and the development of anastomoses. These are (1) the proper technique and (2) the presence of "need" or arterial insufficiency. With attention to these two features, he increased the number of his successful takes from 46 per cent to 75 per cent. Concerning the matter of technique, his descriptions are adequately detailed and lucid. It seems likely that those investigators assessing the operation were able to prepare their animals with satisfactory technical accomplishment. Why they were unable to find patent vessels remains a mystery. It may be noted, however, that those who were able to obtain persistently patent lumens in the implanted vessels followed an operative technique which varied somewhat from that described by Vineberg. Fuquay and associates[5] implanted the left subclavian artery and left its 4 branches open in the myocardial tunnel. Sabiston and associates[12]

made a long tunnel into which they implanted an artery with multiple tangential openings. Smith and associates[14] used a nylon tube to convey the blood from the aorta through a ventricular tunnel, and they likewise had multiple perforations of the implanted portion of the tube. Carey and co-workers[4] anastomosed the end of the implanted artery to the left atrium. The results obtained by these investigators were similar to ours in showing that the majority of the implanted vessels do remain patent. All of these modifications appear to have one thing in common; namely, they are constructed so as to permit a continuous flow of blood through the implanted vessel. It is our opinion that this feature is the critical one in preventing the development of intimal proliferation and obliteration of the arterial lumen.

The second matter to which Vineberg directed attention is the receptive site. A myocardium with reduced arterial circulation, either as the result of surgical ligature or atherosclerosis, is presumably an area which will be more receptive to the blood furnished by an extracardiac vessel. On teleological grounds, therefore, the implanted artery will tend to remain patent and develop anastomoses with the existent arterial tree which is carrying a reduced volume of blood. In Vineberg's reported experiments, it may be noted in the normal hearts that the number of patent vessels was higher than the number with anastomotic connections (65 per cent as compared to 46 per cent) and in the ischemic hearts (76 per cent as compared to 71 per cent). While the latter differences are not significant, it is interesting to note that myocardial ischemia increased the number of patent vessels from 65 per cent in the control group to only 76 per cent. It would seem, therefore, that need is not too important a factor as regards the maintenance of patency. This argument is supported by the reports from other laboratories as well as our own findings that most of the arteries remained patent in hearts without myocardial ischemia.

The point which we wish to emphasize is that the majority of the implanted vessels will remain patent even in normal dog hearts as long as there is significant flow through the vessel. Presumably when anastomoses develop the flow has been adequate. However, the amount of blood flow required for the maintenance of patency is still unknown. Furthermore, the development of anastomotic connections with the coronary circulation does not necessarily indicate that a significant amount of blood is being supplied to the heart muscle. This point is substantiated by Vineberg's work[16, 17] showing a flow of only 6 or 7 c.c. per minute in 2 of 6 dogs despite the existence of anastomoses, the experiments of Sabiston and his co-workers[12] showing an average flow of only 5 c.c. per minute, and by our own observations that the presence of anastomotic connections is not always associated with the delivery of large volumes of blood to the heart. However, Vineberg may be correct in his opinion that implantation of an artery in ischemic heart muscle is more conducive to the development of anastomotic connections between the implanted vessel and the coronary circulation. The modification herein proposed, of implanting the open bleeding end of vessel into the right ventricle, fulfills the need for maintaining flow through the artery while anastomoses are being formed within the myocardium.

SUMMARY

1. The implantation of the left carotid artery in the myocardium of dogs by the standard technique resulted in intimal proliferation and obliteration of the lumen in 89 per cent of the animals by the end of one month.

2. By modifying the procedure so that the distal open end of the implanted artery is placed in the right ventricular cavity, the lumens of 100 per cent of the implanted vessels remained patent, some for as long as a 10-month observation period.

3. The trauma to the implanted artery which results from the repeated myocardial contraction was not responsible for the intimal proliferation.

4. Evidence is presented to indicate that the continuation of blood flow through the vessel is the most important factor in the maintenance of patency. The amount of flow necessary is still not known.

5. The chances for the development of anastomoses between the implanted artery and the coronary arteries appears to be improved in the ischemic heart muscle. However, the demonstration of the presence of anastomotic connections does not indicate that a significant amount of blood is being delivered to the myocardium.

Acknowledgment for valuable assistance is due Drs. John Clift, Bunket Lourvanij, Jerel Katz, and Mr. Earlie Francis.

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THE SURGICAL TREATMENT OF PECTUS EXCAVATUM

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THERE is general acceptance of the rationale for the surgical correction of the abnormality generally referred to as pectus excavatum, particularly when the deformity is marked. There remains uncertainty and disagreement regarding the indications for surgery when the deformity is mild or when the patient's physical development otherwise is unimpeded, and also in regard to the proper age at which surgery should be undertaken. As has been pointed out in a number of publications, the clinical manifestations and the indications for operative correction fall into three categories: cosmetic, functional, and orthopedic or postural, the last category being a function, in part, of the first two.

A number of variations in operative technique have been described since the important publication by Brown,[1] in 1939. These include the two procedures termed "simple" and "radical." The "radical" operation, which consists of the division or resection of the deformed costal cartilages, the removal or the division of the attachments of the xyphoid, and osteotomy of the anterior cortex of the body of the sternum with elevation of the gladiolus, has been modified in many ways since Brown described his operation. Many authors have emphasized the need for fixation of the elevated sternum to an external splint or prosthesis for variable periods of time.[1-5] Others have used methods of internal fixation which employ grafts of rib,[6] or cartilage,[7] or the use of wires[8] and Steinmann pins.[9] Ravitch,[10] in 1949, advocated a procedure in which no traction apparatus or internal splint is used to maintain the elevation of the sternum. He has since then employed such a procedure in a large number of cases with satisfactory results.[11]

Since 1948, we have operated upon 35 patients with funnel breast deformity. Five patients were adults. The youngest was 6 months of age, the oldest 42 years. External fixation by means of wire sutures, as was described by Brown, was utilized in only the first 2. No splinting, either external or internal, has been used in the remainder.

The purpose of this paper is twofold: to add emphasis to Ravitch's contention that external fixation of the mobilized sternum, by means of metal sutures or splints or by grafting, is not needed and to describe a modification of the operation described by Ravitch which has been performed upon 29 patients since 1954.

Received for publication Oct. 7, 1957.
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TECHNIQUE OF OPERATION

The incision may be placed in a vertical direction, extending from the manubrium sterni to a point about midway between the xyphoid process and the umbilicus (Fig. 1, *A-a*), or it may be placed in a transverse manner, in which case it is extended across the xyphoid and to each anterior axillary line (Fig. 1, *A-b*). In the majority of cases we now prefer the transverse incision. The resulting scar of the midline incision may be unsightly and is usually high enough to be exposed through an open collar or low-necked dress.

The skin, subcutaneous tissues, and pectoral muscles are dissected away from the body of the sternum and the costal cartilages to such an extent that the greater portion of the anterior chest wall and the upper parts of the rectus abdominis muscles are exposed. This dissection should be extensive in order that all of the deformed portions of the costal cartilages are exposed. The cartilages are then excised subperichondrially (Fig. 1, *B*). It is important that this include all the deformed cartilages, whether or not the depression is symmetrical. In most cases the resection will include portions or all of the seventh cartilages and progressively shorter segments of the sixth, fifth, fourth, and third.

Following removal of the cartilages, the rectus muscles and the linea alba are freed from the xyphoid and from the perichondrium of the seventh cartilages. The xyphoid is not excised. The anterior cortex of the sternum is then transected at the level at which the posterior angle or curve begins; usually this is at the level of the third intercostal space and a wedge-shaped segment of the bone is excised to permit the sternum to be brought forward.

The sternum is elevated manually and the pleura is freed from the sternal borders and the endothoracic fascia by blunt dissection and is displaced laterally on each side. The parietal pleura on the right side is usually incised purposely to permit drainage into the pleural cavity postoperatively but the pleura on the left side is also often opened inadvertently. Incisions are then extended from the costal margins upward through the entire thickness of the soft tissue flaps, created by the subperichondrial resection of the costal cartilages, in a slightly curving line which is adjacent to the cut ends of the remaining costal cartilages (Fig. 1, *C*). The resulting soft tissue flaps, which have been left attached to the lateral borders of the sternum, are composed of endothoracic fascia, the intercostal muscle bundles, and perichondrium. Care has been taken to preserve the integrity of the internal mammary vessels superiorly, although they are often divided inferiorly as the rectus abdominis muscles are separated from the xyphoid and costal margins.

The soft tissue flaps are then brought outside the rib cage to overlap the costal cartilages and ribs laterally. They are sutured to the fascia overlying the intercostal muscles and to the perichondrium on each side, under moderate tension, with many interrupted sutures of silk (Fig. 2). These flaps, sutured as they are under moderate tension, act as taut "slings" to support the body

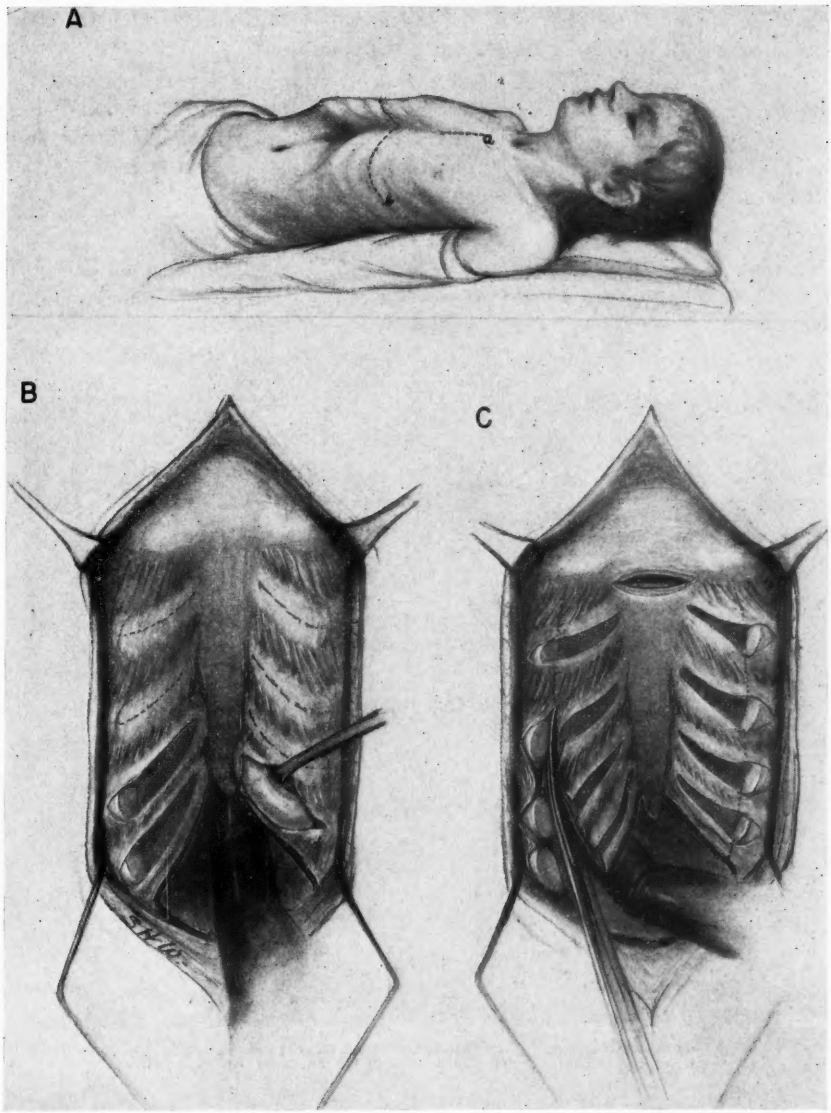

Fig. 1.—*A*, The incision may be in a vertical (*a*) or transverse (*b*) direction. Satisfactory exposure is obtained by either. We now prefer the transverse incision.

B, Excision of the costal cartilages is effected without removal of the perichondrium.

C, The lines of incision through the entire chest wall are depicted, as is the transverse anterior sternotomy.

of the sternum in a normal position. Interrupted sutures of silk are then placed to oppose the cut edges of the fascia and periosteum overlying the sternal osteotomy. No sutures are placed in the bone itself.

The cartilaginous xyphoid, which is sharply angulated forward, is partially divided transversely. It can then be easily molded or fractured so that it lies in a plane with the body of the sternum. The rectus abdominis fascia and linea alba are sutured to the xyphoid and to the lowermost margin of the lateral soft tissue flaps to complete the operation.

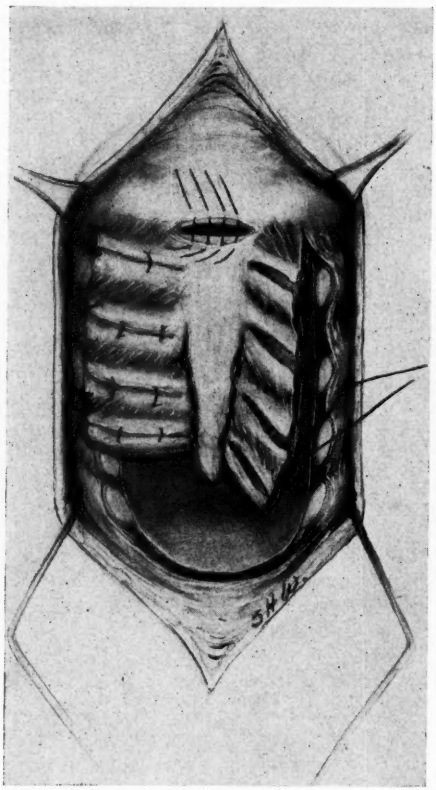

Fig. 2.—The soft tissues, composed of perichondrium, intercostal muscles, and endothoracic fascia, are sutured under slight tension to the chest wall laterally.

If the left pleural cavity has been opened, air is evacuated by means of a small catheter which is removed as the wound closure is completed. An inlying intercostal catheter is left in the right pleural cavity, through a stab wound in the lateral chest wall, for drainage of the mediastinum through the right pleural cavity. The pectoral fascia, subcutaneous tissues, and skin are approximated with fine silk sutures.

DISCUSSION

The results in the patients in whom this procedure has been carried out have been satisfactory on the whole. In the majority the sternum has been maintained in good position. In a few there has been slight retraction of the lower end of the sternum and, in these few patients, this has been noted not during the early postoperative period but always after a period of at least 6 weeks. Thus, the use of an external prosthesis to maintain or "over-correct" the position of the sternum, if removed within the first 2 or 3 weeks after operation, would not have accomplished its purpose.

In a 4-year-old child, on whom the operation employed by Ravitch was carried out, there occurred gradual postoperative rotation of the sternum in such a manner that the right sternal border came to rest in a forward position beneath the skin as a visible and somewhat unsightly ridge or keel. This has not occurred following the technique described herein.

A. *B.*

Fig. 3.—Appearance of anterior chest wall before operation (*A*) and 3 weeks following operation (*B*). Very slight retraction of the lower end of the sternum was noted several weeks later.

The deformities in which this operative procedure has been employed have varied. Usually the curve of the sternal body is concave on its mediastinal surface, while the xyphoid is turned forward at a sharp angle at its junction with the body. Two children, one male and the other female, operated upon by us exhibited a "double curve" of the gladiolus, the anterior being convex as is usual at about the level of the third costal cartilage and slightly concave near its inferior end. Osteotomy was, therefore, performed at the two levels in order to flatten the sternal body and its position has been satisfactorily maintained in both cases. Lester[12] has recently classified deformities of the anterior chest wall and he calls attention to the fact that the position of the deformed

costal cartilages and ribs is quite variable and often asymmetrical. He also notes that occasionally pectus excavatum will be associated with a posterior deformity of the upper end of the sternum and also, probably more frequently, with a flaring of the lower costal cartilages and ribs anteriorly. The unsightly flaring of the lower cartilages can often be improved or corrected entirely by the complete excision of not only the depressed medial ends of the lower cartilages but the flared lateral portions as well.

The operative procedure herein described has been carried out effectively and with considerable improvement, from a cosmetic standpoint, in one patient, a 16-year-old boy, with a marked protrusion deformity (pectus carinatum) of the sternum and anterior chest wall. Pulmonary function studies, carried out prior to and some weeks following the operation, revealed no change.

Pulmonary function has been measured in a number of children with pectus excavatum before and after operation. A number of these are still undergoing study. The results of these tests will be published later, but it does appear now that a definite increase in the pulmonary function, as related to the growth of the children, is measurable during periods of time which vary from a few weeks to several months following surgical correction of the deformity.

The value of the operative procedure from a functional standpoint has been well demonstrated in 3 children with cerebral palsy. Four such children, all with marked sternal depression and inspiratory chest wall retraction, have been subjected to operation. One died following cardiac arrest during operation. The remainder have been almost completely relieved from frequently recurring severe respiratory infections which had added much to the difficulty of their care prior to the corrective operations.

There have been no other deaths following operation except for the one recorded above. Complications have been few. One child, a 7-year-old girl, with cerebral palsy, suffered two prolonged generalized convulsive seizures on the first postoperative day. Her subsequent recovery was uncomplicated. Aspiration of small amounts of serous fluid from the wounds was necessary in 3 patients. There were no wound infections. The average time of hospitalization after operation has been 7 days.

SUMMARY

An operation for the correction of developmental deformities of the anterior chest wall is described in detail. The procedure involves an extensive dissection and mobilization of the sternum and the adjacent anterior chest wall and no external prosthesis or splint is employed. The postoperative care is simple. Complications have been few and the total period of hospitalization rather short. The procedure has been carried out on 28 patients with varied types of funnel chest deformity and on one patient with pectus carinatum or pigeon-breast. The results, from both the functional and cosmetic standpoint, have been satisfactory.

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DIRECT LYMPHATIC CONNECTIONS FROM THE LOWER LOBES OF THE LUNG TO THE ABDOMEN

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INTRODUCTION

ABDOMINAL metastasis is relatively common in cancer of the lung. In the 3,047 collected cases of pulmonary cancer studied by Ochsner and DeBakey,[10] 78 per cent were metastatic to mediastinal lymph nodes, 33 per cent to the liver, 20 per cent to the adrenals, and 17 per cent to the kidneys. In 20 per cent of the 1,298 cases in which such information was recorded, metastasis to abdominal lymph nodes occurred. Although spread to abdominal lymph nodes is patently lymphatic, dissemination to abdominal organs may be by way of the lymph or blood vessels, by direct extension of the tumor, or by any combination of these. We still do not know which route of spread is most important and at what point, in the course of pulmonary carcinoma, metastasis occurs, despite the importance of these to therapy of the disease.[6]

Metastasis to abdominal nodes is usually considered to result solely from anomalous drainage caused by neoplastic invasion or adhesions of the lung to parietal or diaphragmatic pleura. However, Rouviere[11] described 2 specimens, and Mouchet and Nouriddine[9] one, in which the lymphatic vessels coursed from the lower lobes to a retroesophageal diaphragmatic node whose efferent ended in an abdominal para-aortic node. De Sousa,[5] in 2 specimens, found vessels from the left lung that ran without interruption to nodes of the gastric cardia or abdominal para-aortic nodes. In one of these, a vessel, which left the mediastinal surface of the right lower lobe, passed through 2 paraesophageal nodes before ending in an abdominal para-aortic node. This mode of lymph drainage could explain some of the cases of metastasis from pulmonary cancer to abdominal lymph nodes, but none of these authors seemed to consider it as other than an uncommon variant, and the existence of such a vessel is, in fact, specifically denied in a recent textbook of anatomy.[8] For these reasons, the possibility of such spread is seldom considered in the planning of therapy for patients with cancer of the lung.

The failure of more frequent demonstration of such routes of intra-abdominal pulmonary lymphatic drainage is surprising in view of the embryologic development of pulmonary lymphatic vessels. Cunningham[4] demonstrated that the pulmonary lymphatic vessels arise from 3 sources: the right and left jugular lymph sacs and the retroperitoneal lymph sac. The vessels from the jugular

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Presented at the meeting of The American College of Chest Physicians in Miami, Nov. 9-11, 1957.
Received for publication Oct. 16, 1957.

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lymph sacs, which lie in the cervical region, grow downward along the trachea to the lungs. The left grows faster than the right and joins with it at the tracheal bifurcation, sending a large contribution to the right lung as well as to the left. From the retroperitoneal lymph sac, which lies in the upper part of the abdomen, arises the lymphatic supply of the lower third of the esophagus, the stomach, pancreas, spleen, liver, adrenals, and kidneys as well as the diaphragm and lower third of each lung. Usually 3 or 4 vessels, closely associated with those of the diaphragm, arise from the cephalic portion of the sac and grow dorsal to the stomach into the lung (Fig. 1). The vessels to the diaphragm supply that structure, and the pulmonary lymphatics extend into the pulmonary ligament about one third of the way to the hilum, dividing there into 2 branches.

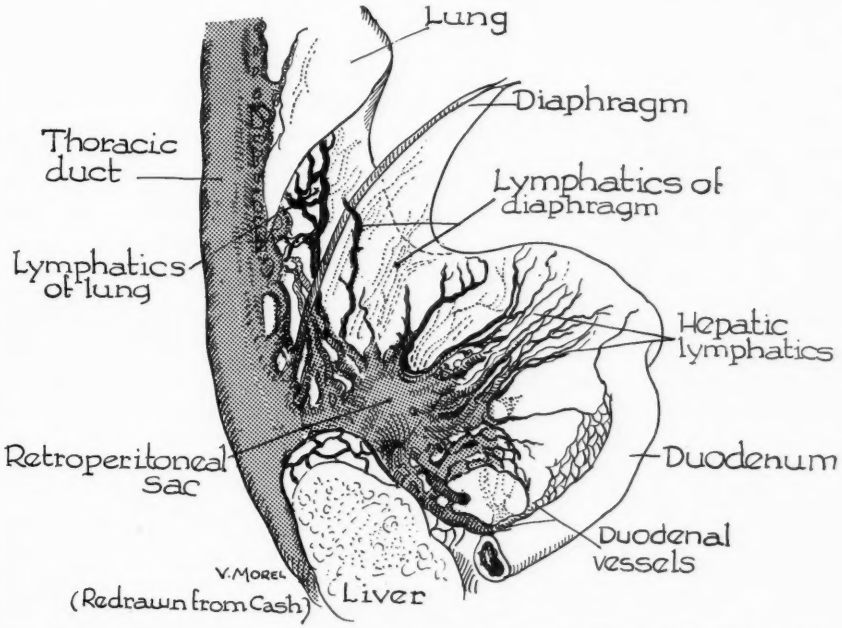

Fig. 1.—Diagrammatic representation of lymphatic vessels arising from the retroperitoneal sac, from Cash.[2] The lung and diaphragmatic lymphatic vessels originate in common and, as the latter spread over the diaphragm, the pulmonary lymphatics pass through it to reach the lower third of the lung. Lymphatic vessels to the upper abdominal viscera also arise from this sac.

One supplies the lateral and the other the diaphragmatic surface of the lower lobe. These grow into the lung and join with those extending out from the hilum.

Since the vessels from the right and left lymph ducts persist in the adult as the right and left tracheobronchial chain, emptying into the respective jugulosubclavian trunks, it seems likely that the vessels from the retroperitoneal sac would also remain. If they do, and if flow through them to the abdomen is not impeded by valves, this mode of pulmonary lymph drainage would, as

Cunningham[4] suggested, have considerable clinical significance. Although previous studies indicate only rare persistence of this fetal state, the poorer prognosis of cancer of the lower lobe[1] (13 per cent of patients with cancer of the lower lobe survive 2 years as compared with 22 per cent of those with carcinoma of the upper lobe) and the extensive spread of pulmonary carcinoma to the upper part of the abdomen suggest that such vessels exist commonly. The purpose of the present study was to determine the status of these channels, the extent to which they persist, and their character.

MATERIALS AND METHODS

Twenty-three stillborns and neonates, which were anatomically normal, were obtained after storage in a morgue refrigerator for 72 hours. In some, injection and study were done immediately but most were preserved by freezing at −10° F. for several days to months. This freeze storage had no noticeable effect on the injection.

Since lymphatic vessels are more successfully injected when the tissues are warmed,[14] all specimens were thawed and immersed in warm water to bring them as closely as possible to body temperature. The ribs were then removed to obtain good exposure of the lungs but the sternum and costal arches were left in place to support the mediastinum and diaphragm in correct anatomic relationships. After the lungs had been inflated through a tracheostomy, India ink, diluted with 3 parts of distilled water, was injected subpleurally through a 26-gauge needle, according to the technique of Simer.[14] The lower third of the respective lower lobe, the region that receives the vessels from the retroperitoneal sac in the embryo, was injected carefully with particular attention to the diaphragmatic and medial surfaces near the pulmonary ligament. This area is difficult to inject because the pulmonary ligament must not be stretched or twisted during the injection. If it is, the vessels that course in its lower free edge may be blocked and thus not visualized; these vessels course to the abdomen.

In successful injections, the pleural lymphatic network was filled in a short time and the ink flowed rapidly within the pleural collecting vessels. The lung was then gently stroked to aid the flow of ink along the vessels. Usually only 1 c.c. of ink was injected as this gave adequate filling of lymphatics and larger amounts disrupted the substance of the lung. The ink-filled vessels that left the lung were traced with blunt and sharp dissection, loupe binoculars or a dissecting microscope being used. Whenever possible, the node into which the vessel drained was also injected. However, the small size of these nodes made this most difficult and was usually unsuccessful. The specimens were then preserved in 10 per cent formalin for further study; sketches of the course of the ink-filled vessels were made.

OBSERVATIONS

The lymphatic vessels in the right lower lobe were demonstrated in 15 specimens and those in the left in 12 specimens. The criterion of successful injection was the filling of lymph vessels that emptied into tracheobronchial

nodes and all specimens displayed drainage to this area through the usually described channels. Only those vessels that coursed to the abdomen will be described.

When present, these vessels arose from the diaphragmatic surface or the lower medial surface of the lower lobe. Within the pulmonary ligament, they invariably coursed near its lower free edge, following the ligament to its attachment to the diaphragm. Usually the several small vessels that left the lung united into a single trunk. This continued as a single trunk or it divided into two or more channels. These then passed to the abdomen without interruption, or joined the infrapericardial nodes or the posterior diaphragmatic lymphatics before terminating in the upper part of the abdomen.

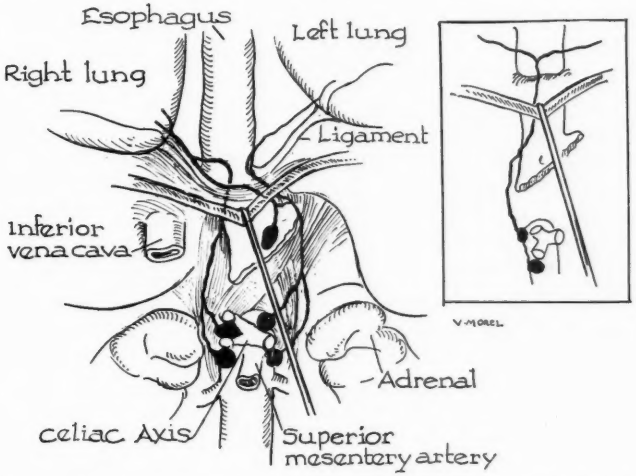

Fig. 2.—Diagrammatic representation of pulmonary lymphatic vessels that drain to the celiac nodes. These vessels usually pass through the esophageal hiatus, then run along either crus to the aortic nodes near the celiac axis. They may pass directly through the diaphragm. Occasionally they terminate in or send a branch to a node of the cardia. The inset shows the union of vessels from the right and left lungs observed in one specimen.

In Fig. 2 is shown the general path of those vessels that coursed uninterruptedly to abdominal nodes. These were observed issuing from the right lower lobe in 2 of the 15 successful injections of this lobe and from the left lower lobe in 3 of 12 successful injections. The vessels leaving either lobe passed in the pulmonary ligament to reach the esophagus slightly above the hiatus or ran on the diaphragm for a short distance before traversing the hiatus anterior, posterior or lateral to the esophagus. In one specimen the vessel from the left lower lobe passed through the diaphragmatic musculature instead of the hiatus. Within the abdominal cavity the vessels left the esophagus to run down either crus to the nodes about the celiac axis. Some terminated in, or sent a branch to, the nodes of the cardia before ending in the celiac nodes.

The inset in Fig. 2 shows the union of the vessels from the right and left lower lobes which was observed in one specimen. These vessels joined anterior

to the esophagus and above the diaphragm. A single trunk coursed through the hiatus to the right of the esophagus and then ran down the abdominal surface of the right crus to end in 3 nodes about the celiac axis.

Vessels that joined the diaphragmatic nodes were observed in 6 specimens of the right lower lobe and in one of the left lower lobe. These nodes and their connections are illustrated in Fig. 3. Because these nodes are often so small,

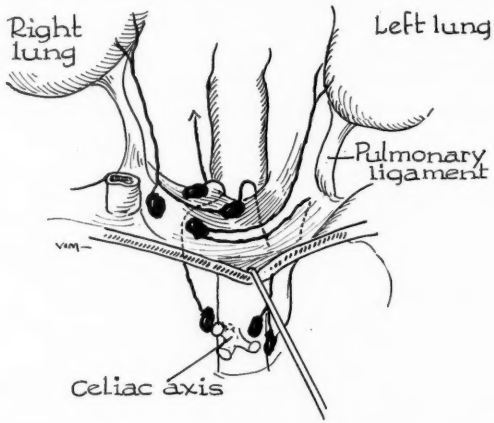

Fig. 3.—Diagrammatic representation of the infrapericardiac nodes and their connections. These lie between the pericardium and diaphragm and receive lymph from the liver as well as the lungs. Their efferents are to abdominal peri-aortic nodes, nodes of the cardia and carinal nodes. Lymph from the right lung drains to these nodes more often than does that of the left.

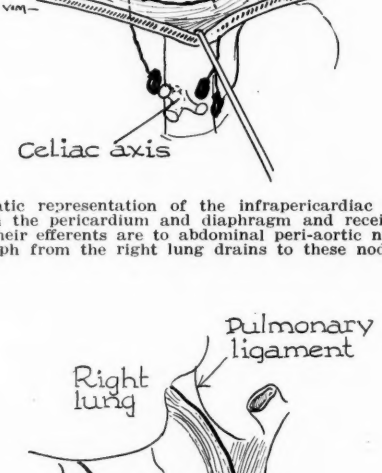

Fig. 4.—Diagrammatic representation of the pulmonary lymphatic vessels that join the posterior diaphragmatic lymphatic vessels. These were observed from the right lung only. The diaphragmatic vessels, in turn, join those of the liver and adrenal before emptying into the para-aortic and celiac nodes.

it was not always possible to trace their efferent connections. However, those demonstrated ascended to tracheobronchial nodes or coursed backward to a retroesophageal node or passed through the diaphragm to nodes about the celiac axis.

The general course of the vessels that ran on the posterior diaphragm is shown in Fig. 4. These vessels were demonstrated in 4 specimens, all from the right lower lobe. As they coursed posteriorly or posterolaterally, their branches joined with those of the intrinsic diaphragmatic lymphatics.

DISCUSSION

Direct lymphatic communication between the lung and the upper abdominal viscera and lymph nodes is either by way of vessels that pass through the esophageal hiatus to the celiac nodes or by confluence of lymph vessels from the lung with those of the posterior diaphragm. Although the vessels that pass through the esophageal hiatus may have no direct communication with abdominal organs other than the stomach, those blending with the posterior diaphragm are, through its lymphatics, in direct continuity with the vessels of the liver, adrenal gland, and kidney before draining ultimately to the celiac and para-aortic lymph nodes.

From both lower lobes, the subpleural collecting vessels that course directly into the abdomen arise from or just above the diaphragmatic surface of the lung and run in the pulmonary ligament near its lower free edge. Traveling medially, they reach the esophagus slightly above the hiatus. Then, paralleling the esophagus, they traverse the hiatus anterior, posterior, or lateral to the esophagus, either separately or as a single trunk resulting from the union of the lymphatics from each lung. Within the abdominal cavity the vessels leave the esophagus to run down the abdominal surface of either crus, frequently skirting the adrenals, and end in the nodes about the celiac axis and aorta.

The diaphragmatic nodes frequently interposed in the course of the vessel from the right lung, and occasionally from the left, lie near the esophageal hiatus, to the left of the inferior vena cava. These nodes, the infrapericardial nodes, which were described and named by Sledziewski,[15] vary in number (from 1 to 5) and size but always lie sandwiched between the pericardium and the diaphragm and thus are not readily visualized. As did Sledziewski, I found multiple efferents from these nodes. Some ascend to tracheobronchial nodes; others pass backward to retroesophageal nodes from which they may empty into the thoracic duct or to intra-abdominal para-aortic nodes.[9, 11] Others pass through the esophageal hiatus or diaphragm to end in nodes of the cardia or celiac axis and so carry pulmonary lymph to the abdomen.

Of great importance as a route of metastasis of pulmonary tumor to abdominal viscera are those pulmonary lymphatic vessels that blend with the intrinsic lymphatic vessels of the diaphragm. These vessels, which reach the diaphragm through the pulmonary ligaments, and then pass posteriorly or posterolaterally on the diaphragm, were found only from the right lung; this confirms the observations of Sappey[13] and Cruikshank.[3] Their importance is that the lymphatic network of the posterior diaphragm joins not only both diaphragmatic surfaces but some of the lymphatics draining the adjacent viscera—lungs, liver, adrenals, and kidneys—into a common interconnecting pool, which drains eventually to nodes about the upper end of the abdominal aorta.[12]

In describing the lymphatic drainage of the diaphragm, Rouviere[12] emphasized that its extensive subpleural and subperitoneal lymph networks are intimately connected by penetrating vessels, which drain to the subpleural collecting trunks. In the posterior half of the diaphragm, these pass through the diaphragm to end in nodes about the cardia, the upper part of the abdominal aorta and the origin of the inferior phrenic arteries. These nodes are the ones into which the uninterrupted channels from the lung empty.

In addition, Rouviere stressed that some of the lymphatic vessels of the liver and adipose capsule of the kidney and adrenal ascend to penetrate the diaphragm and join the subpleural collecting trunks while others join the collecting trunks of the diaphragm which course along the inferior phrenic artery. The continuity of these collecting trunks with the adrenal lymphatics is well illustrated in Higgins and Mills'[7] physiologic study of absorption from the peritoneal cavity.

The present study has demonstrated that the diaphragm is not a barrier to, but rather a junctional link between, the lymphatic vessels draining the thoracic and upper abdominal viscera and that, in the absence of adhesions between parietal and diaphragmatic pleura, there are routes through which pulmonary lymph drains to abdominal nodes.

The role of this functional continuity in the metastatic pattern of cancer of the lung is readily apparent. Since the celiac and para-aortic lymph nodes are the first echelon of nodes of pulmonary lymph drainage, a tumor of the lung can spread to the abdominal lymph nodes as readily as to mediastinal nodes—and this spread may not be evident at thoracotomy. Through the blending of lymph from the lungs, liver, adrenals, and kidneys in the posterior diaphragm, neoplastic cells within the diaphragmatic lymphatic vessels can spread directly to these organs. Metastatic tumor from the lungs, growing in celiac or para-aortic lymph nodes, can pass within the afferent vessels of these nodes to any of the upper abdominal organs (the reverse may also occur). Lymph-borne abdominal metastasis in cancer of the lung may occur early and must be suspected in every patient with cancer of the lower lobes of the lung.

SUMMARY

The lymphatic drainage of the lower lobes of the lung was studied to determine if any lymphatic vessels from the lung course directly to the upper part of the abdomen. Twenty-three stillborns and neonates were preserved by refrigeration until used, when they were warmed and the lungs were inflated. India ink, diluted 1:3 with distilled water, was injected subpleurally into the mediastinal and diaphragmatic surfaces of the lobes and the ink-filled vessels were traced to their terminations. The anatomic relationships of the pulmonary ligament were carefully maintained during injection.

Eighteen specimens were successfully injected. The lymphatic vessels of the right lower lobe were demonstrated in 15 of these and of the left lower lobe in 12.

Vessels from the right lower lobe that coursed to the celiac nodes without interruption were observed in 2 specimens. They entered the abdomen by passing through the esophageal hiatus and then ran on either crus to the nodes about the celiac axis. Other lymphatic vessels from the right lower lobe, which ran in the pulmonary ligament to the diaphragm, were demonstrated in 9 specimens. On reaching the diaphragm they either joined with the lymphatic vessels of the posterior diaphragm or ended in a pre-esophageal diaphragmatic node. Some of the efferents of this node group ended in nodes about the celiac axis.

From the left lower lobe, vessels that coursed to the abdomen were observed in 3 specimens. They ran medially in the pulmonary ligament to the esophagus, entered the abdomen by traversing the esophageal hiatus and ran on either crus to nodes about the celiac axis.

The communications of the lymph vessels of the posterior diaphragm with those of the lungs, liver, adrenals, kidneys, and abdominal lymph nodes have been briefly reviewed.

From these observations the conclusion is drawn that pulmonary lymph normally drains to the abdomen and that early, direct metastasis of pulmonary cancer to the abdomen, through the vessels demonstrated, may account for the frequency of intra-abdominal metastases in pulmonary cancer.

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SURGICAL REPAIR OF A TRAUMATIC AORTIC-RIGHT VENTRICULAR FISTULA

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AORTIC-RIGHT ventricular fistula is a rare cardiac lesion. Sawyers and his associates[1] recently reviewed the literature since 1840, and found only 19 cases. Since that report, a few instances in which surgical closure was attempted or carried out have been reported.[2, 3, 4]

Aortic-right ventricular fistulas usually occur following rupture of an aneurysm of the right coronary sinus into the ventricular chamber. Aneurysms of the sinus of Valsalva are most frequently of congenital origin, although some have occurred following syphilitic or bacterial infection. In the absence of endarteritis, no symptoms occur until the aneurysm ruptures. At that time, there is usually chest pain and difficulty in breathing.[5, 6] Often, a whirring sensation is felt in the precordial region. Over the next few days the symptoms generally decrease in their intensity. The patient then enters a temporary, relatively symptomless, phase. The average life expectancy following development of a fistula, however, is usually about one year. Death most frequently occurs following congestive heart failure. In a few cases, bacterial endocarditis develops and death ensues from an overwhelming bacteremia. In the average case, death occurs in the third decade of life.

Trauma must be a most unusual cause of aortic-right ventricular fistula. Interventricular septal defects and aortic-pulmonary fistulas have occurred infrequently following cardiac trauma. Gerbode[7] reported a case of traumatic aortico-atrial fistula caused by a bullet. We have been unable to find the record of any case of traumatic aortic-right ventricular fistula.

This report is concerned with an aortic-right ventricular fistula which occurred following a gunshot wound of the chest. The eventual clinical diagnosis was confirmed by cardiac catheterization, cineradiography, and by exploration of the right ventricle at the time of surgical repair.

CASE REPORT

The patient was a 15-year-old white boy who was first admitted to the Indiana University Medical Center on Sept. 23, 1956. On the morning of admission, the patient was hammering a blank 30-30 cartridge when it exploded and struck him in the chest. He was severely dyspneic on arrival at his local hospital. A loud cardiac murmur was heard. He was transferred to the Medical Center 10 hours after injury. At that time, his pulse was 140 and regular, respirations 22, and temperature 100.8°. The blood pressure was recorded as 120/0 mm. Hg on one occasion and 120/30 on another. After the first 4 hours of hospitalization, the diastolic pressure gradually increased and the blood pressure ranged from 110/40 to 118/60 mm. Hg. The pulse remained at 140 to 150 per minute. He was in acute

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Aided by grants from the James Whitcomb Riley Memorial Association and the United States Public Health Service.
Received for publication Oct. 30, 1957.

distress. In the third interspace, about 5 cm. from the midline, there were two small lacerations. A small amount of subcutaneous emphysema was present over the left upper chest. A harsh, Grade 4 to 5, continuous murmur was heard throughout the entire precordial area but most loudly in the second left anterior interspace, approximately 1 to 2 cm. to the left of the midsternal line. A continuous thrill was palpated in the same area. The hemoglobin determination and erythrocyte count were normal and there was a slight leukocytosis. Urinalysis showed a reducing substance to be present in the urine; however, a glucose tolerance test done later was within normal limits. Cardiac fluoroscopy revealed a slight increase in the size of the pulmonary conus and a questionable enlargement of the left ventricle. The pulmonary arteries were prominent, but the peripheral lung fields were fairly clear. An irregular, opaque foreign body, measuring 1 cm. in greatest diameter, was present in the left anterior mediastinum (Fig. 1). On lateral view, the foreign body was seen just beneath the sternum.

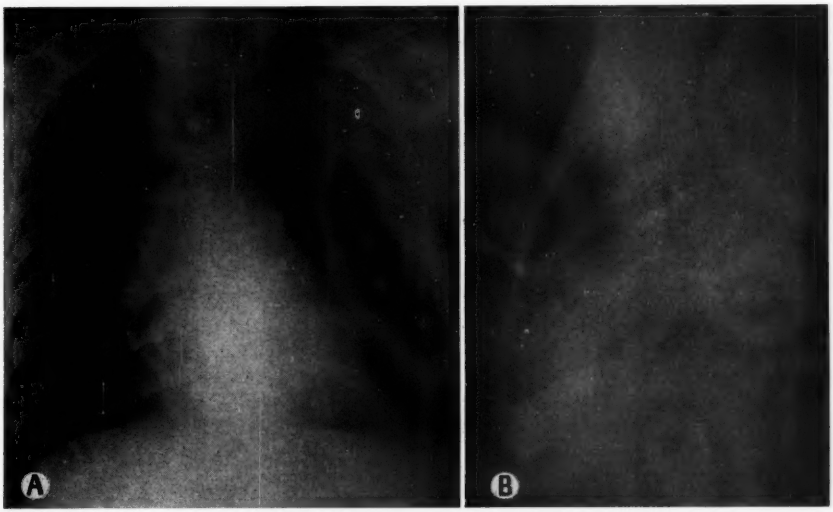

Fig. 1.—Roentgenograms taken at the first admission.

During the 4 days after admission, the patient's rapid pulse rate gradually slowed to an average of 85 per minute. The blood pressure stabilized at about 100/50 to 100/60 mm. Hg. The low-grade fever present on admission also subsided. The patient's dyspnea disappeared. Our initial impression was that the continuous murmur resulted from the injury to the chest. Later, however, we arrived at the erroneous conclusion that the patient more likely had a congenital patent ductus arteriosus, and that the injury was unrelated. The site of injury healed and the patient was discharged.

He was readmitted on December 4. The blood pressure was 95/50 mm. Hg and the pulse was 92 per minute. The same cardiac findings were present. On consultation the pediatric cardiologist still felt that the patient had a typical patent ductus arteriosus. Therefore, on December 6, the patient's left thorax was explored. A patent ductus was not present. The continuous thrill could be palpated through the pericardium anteriorly in the region of the base of the pulmonary artery or right ventricular outflow tract. In this area there was dense scar tissue and adhesions of the pericardium to the anterior chest wall. Since it seemed likely that open cardiotomy with bypass of the heart and lungs would be necessary to cure the lesion, the pericardium was not opened at this time and the chest was closed. The patient's postoperative course was good and he was discharged from the hospital.

While home, he did not do well. He had frequent attacks of pulmonary congestion. Some were thought to be upper respiratory infections but others were clearly due to cardiac failure. Because his physical condition seemed to be gradually deteriorating, he was readmitted on March 9, 1957. Physical examination was the same as before. The blood pressure varied between 114/50 and 110/60 mm. Hg. The pulse was 90 per minute. On cardiac catheterization, oxygen saturation of the blood increased from 79 in the atrium to 95 per cent in the region of the right ventricular outflow tract. The pulmonary artery and right ventricular pressures were not increased, both being around 26 mm. of mercury. Cineradiography demonstrated dilution of the opaque media in the outflow tract of the right ventricle and this presumably was due to a left-to-right shunt at this point. The fistulous tract itself was not seen. These data demonstrated a left-to-right shunt into the right ventricle.

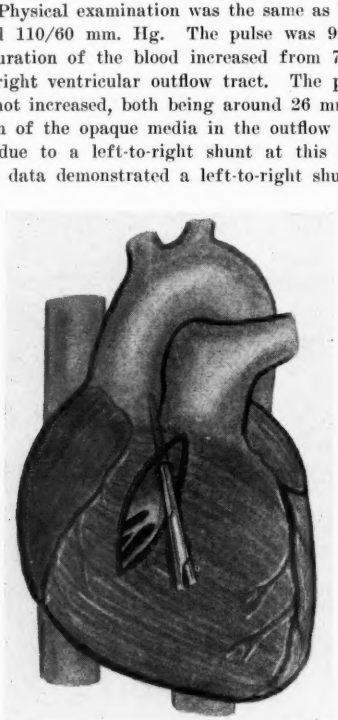

Fig. 2.—Drawing showing the fistulous tract as observed through the right ventriculotomy incision. A large clamp has been placed through the tract into the aorta. At operation, the cardiac landmarks were obscured by the overlying pericarditis.

They did not permit a clear differentiation between a ventricular septal defect and an aortic-right ventricular fistula. Taking everything into consideration, we reached the conclusion that the best tentative diagnosis was a fistulous tract between the right sinus of Valsalva and the right ventricular outflow tract.

On March 13, a bilateral anterior thoracotomy was carried out in the fourth interspace. The sternum was split transversely. Before operative repair, the blood pressure ranged from 130/60 to 140/70 mm. Hg. The pulse rate was 80 per minute. There were numerous adhesions between the anterior chest wall and the pericardium. The pericardium was adherent to the heart and great difficulty was encountered in dissecting it free. Even after this was accomplished, it was impossible to recognize superficial cardiac landmarks such as the coronary arteries. Only the general outline of the right ventricle could be defined. The point of maximum thrill was located about one inch below the base of the aorta over the right ventricular outflow tract. Here, recent scar tissue, about 0.5 cm. in diameter, protruded from the ventricular wall. The venae cavae and the left femoral artery were cannulated and cardio pulmonary bypass was begun at the rate of 35 c.c. per kilo per minute. Twenty-five per cent potassium citrate solution dissolved in blood in a ratio of 2 c.c. to 18 c.c. was injected into the base of the aorta after it was clamped. Over 100 c.c. of this solution were required to arrest the heart. In retrospect, a substantial amount of the potassium citrate probably went through the fistulous tract into the right ventricle instead of entering

the coronary arteries. After cardiac standstill was achieved, the right ventricle was opened through a vertical incision made downward from the base of the pulmonary artery. The fistula was easily seen (Fig. 2). It measured about one centimeter in diameter. A narrow fibrous band lined the edge of the defect. A Kelly clamp was easily passed through this opening and emerged within the aorta in the region of the right coronary sinus of Valsalva. The defect was closed with a running suture of 5-0 silk and reinforced with 2 interrupted 5-0 silk sutures. The right ventriculotomy was then closed. After the clamp was removed from the aorta, ventricular fibrillation began and four electric shocks were necessary to convert the heart action to a normal rhythm. A blood pressure of 120/90 to 130/90 mm. Hg was obtained almost immediately. The pulse was 100 per minute. After the cannulas were removed, a large part of the scarred anterior pericardium was excised.

Fig. 3.—Roentgenogram of chest taken a few weeks after operative repair.

The patient's immediate postoperative course was good. The blood pressure ranged from 130/80 to 120/80 mm. Hg with a pulse rate of 120 per minute. Two days after operation, he had a generalized convulsion involving all extremities and was mentally confused. The serum sodium was found to be 123 mEq. with a blood pH of 7.64. The electrolyte abnormalities were corrected over the next 2 days and, on March 17, he was coherent and rational. He was discharged from the hospital 15 days after operation in good condition.

The patient gained 13 pounds in the first 8 weeks after operation. His exercise tolerance greatly increased. Seven months after operation, the blood pressure varied from 118/70 to 105/68 mm. Hg with a pulse rate around 85. The cardiac sounds were good and no thrill or murmurs were present. A roentgenogram of the chest showed the heart to be normal in size and configuration (Fig. 3). Fluoroscopy of the patient's body revealed a thin metallic fragment, measuring about 1 by 1.5 cm. in size, located about 3 cm. to the left of the first sacral vertebral body (Fig. 4). This fragment undoubtedly created the cardiac fistula and then passed into the aortic stream as an embolus to lodge in the left common iliac, external iliac, or hypogastric artery. The arterial pulsations in the left leg were excellent. It is planned to remove the metallic fragment in the near future.

DISCUSSION

The aortic-right ventricular fistula, in this case, was secondary to the perforating wound of the left anterior chest. Until the metallic embolus was discovered by fluoroscopy and x-ray study, the mechanism of the injury was puzzling. It now seems clear that the fragment penetrated the right ventricular wall at the point where the superficial scar tissue was evident on exploration and then passed through the underlying ventricular septum directly into the aorta and from there distally into the iliac arterial bed. Whether it entered the aorta through the main right coronary artery or through the wall of the right coronary sinus of Valsalva remains a question, though in view of the relatively large size of the foreign body and of the fistulous tract the latter seems much more plausible. A clamp could be passed easily through the fistula into the aortic sinus. It was concluded that it probably connected the sinus and the ventricular chamber directly.

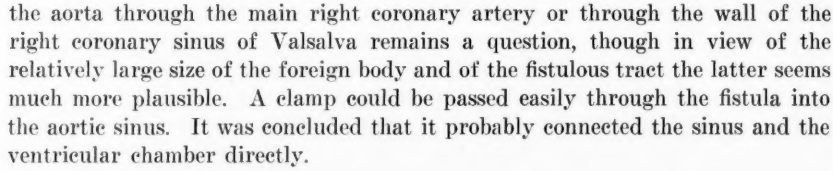

Fig. 4.—Roentgenograms of the pelvis show an embolic metallic fragment located to the left of the first sacral vertebra.

The course of the patient's blood pressure is of interest. Shortly after injury the pulse pressure varied from 90 to 120 mm. Hg. This gradually decreased but remained greater than normal. During anesthesia and before operative repair, the pulse pressure was 70 mm. Hg. Immediately after operative repair, the diastolic pressure increased and the pulse pressure fell to from 30 to 40 mm. Hg. Since recovery from operation, pulse and blood pressure have been normal. Altogether his pulse and blood pressure changes are those which might be expected from any vascular fistula between a high and a relatively low pressure area.

Any aortic-right ventricular fistula, regardless of etiology, would probably produce the same clinical picture as that described following rupture of an

aneurysm of the sinus of Valsalva into the right heart. In most cases, if the patient survives the initial insult, there is a period of a few weeks to a few months during which he remains in relatively good health. It is during this time that surgical therapy can best be carried out. Later, the patient tends to become progressively more disabled and the risk of operation undoubtedly increases. If surgery is deferred until after bacterial endocarditis has become well established, the risk will be even greater.

The surgical technique for closure of these defects is not yet well established. Our patient, treated by suture closure of the right ventricular portion of the fistula, has done well. Others have reported success with the same technique. In dealing with a coronary sinus-right atrial fistula, Baker, Hanson, and Morrow[8] plugged the fistulous tract with molded Ivalon sponge. They feel that this technique will more effectively prevent recurrence of the lesion by at least partially obliterating the aneurysm. Ventricular suture of the defect has given good results in our case and in several others. Although it would have been easy to have inserted a piece of Ivalon sponge into the fistulous tract in our patient, we decided against this maneuver since we could not definitely rule out involvement of the proximal portion of the right coronary artery and did not wish to risk occluding it.

SUMMARY

No case of aortic-right ventricular fistula of traumatic origin has been found in the literature. A case report is presented in which a 15-year-old boy sustained a gunshot wound of the chest resulting in penetration of the right ventricle by a metallic body which passed into the aorta, producing a fistulous communication between the two. All the signs of a patent ductus developed including those of cardiac failure. At operation, using a cardiac bypass, the right ventricle was incised and the opening closed with complete recovery.

We wish to express our appreciation to Dr. Paul Lurie who performed the catheterization studies and to Dr. Eleanor Clay who referred the patient for treatment. We are very grateful to Dr. Emile Holman who suggested the possibility of a second metallic foreign body which had caused the cardiac injury and passed into the distal arterial circulation.

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INTRACARDIAC PRESSURE MEASUREMENT DURING OPERATION FOR CONGENITAL HEART DISEASE

Value of the Method for Assessing the Effect of the Operation

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INTRODUCTION

During the year 1954 an increasing number of patients with valvular pulmonary stenosis without aortic overriding were operated upon at the surgical department of our hospital. The operation was performed transventricularly, according to Brock,[2] so that the valvulotomy took place without direct control of the eye. In these circumstances, the idea of being able, at least indirectly, to observe the result of the splitting of the valve in the course of the operation seemed attractive.[4]

With this end in view, attempts had been made in various quarters to get some idea of the reduction in pressure in the right ventricle in the course of operation. This was done with isolated pressure recordings, either via a puncture needle through the wall of the right ventricle, or with the help of a catheter introduced into the right ventricle through the operator's incision in the heart wall.

Pressure measurements via a catheter introduced into the heart before the operation seemed to me more advisable from several points of view.

1. The pressure values could then be obtained at any time—immediately before, during, or immediately after the operation, and not only during the phase in which the heart was directly accessible for the surgeon's manipulations.

2. Further damage to the ventricular wall through the puncture could thus be avoided.

3. The operator and his assistants need not be disturbed by interference in the pressure-measurement procedure.

4. It would be of great interest to obtain, during certain stages of the operation such as the valvulotomy, *continuous pressure recordings* from the right ventricle.

METHOD

Before the operation, while the patient is still conscious and has been given only the usual premedication (morphine scopolamine), a catheter (Cournand No. 7 or 8) is introduced via the cubital vein or vena saphena magna and disposed with the point in the desired position, as a rule the right ventricle. The child is then transferred to the operating theater, where the catheter is connected

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Received for publication Nov. 19, 1957.

with a pressure-measuring apparatus (Electromanometer, Elema) coupled to a direct-recording, 2-lead electrocardiogram apparatus (Mingograph 23, Elema). When the operative wound has been closed, the catheter is removed, generally about 6 hours after its introduction into the heart.

Postoperative examination, including cardiac catheterization, is carried out not earlier than one month after the operation.

The peripheral blood pressure is measured bloodlessly with a sphygmographic cuff coupled to a mechanical manometer.

The pulse rate is calculated from the ECG-curves.

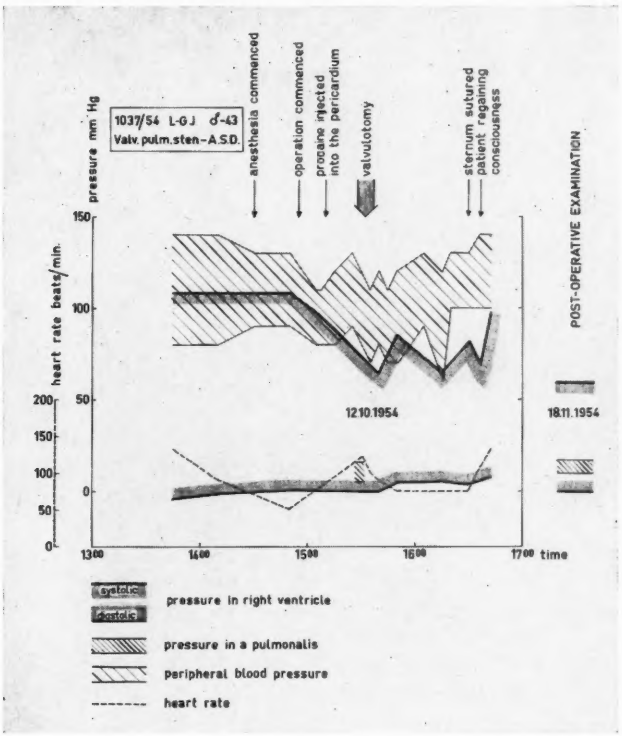

Fig. 1.

MATERIAL

The pressure measurements have been carried out in this way during the operation for 5 valvular pulmonary stenoses, 1 atypical patent ductus arteriosus, and 1 case of coarctation of the aorta. The recordings from 2 patients with pulmonary stenosis were technically unsatisfactory; the pressure values in the other cases are represented graphically in Figs. 1-5.

Fig. 1 shows the recordings of pressure changes in the right ventricle in connection with valvulotomy performed on an 11-year-old boy with valvular

pulmonic stenosis combined with a slight defect in the auricular septum. Even before the start of the valvulotomy, the pressure sank from 110 to 80 mm. Hg after the inception of narcosis. During the splitting of the valve, the pressure dropped somewhat further, to show varying values as the patient began to regain consciousness.

The postoperative examination one month later shows, in the waking state, a pressure lower than the lowest pressure measured in the course of the operation.

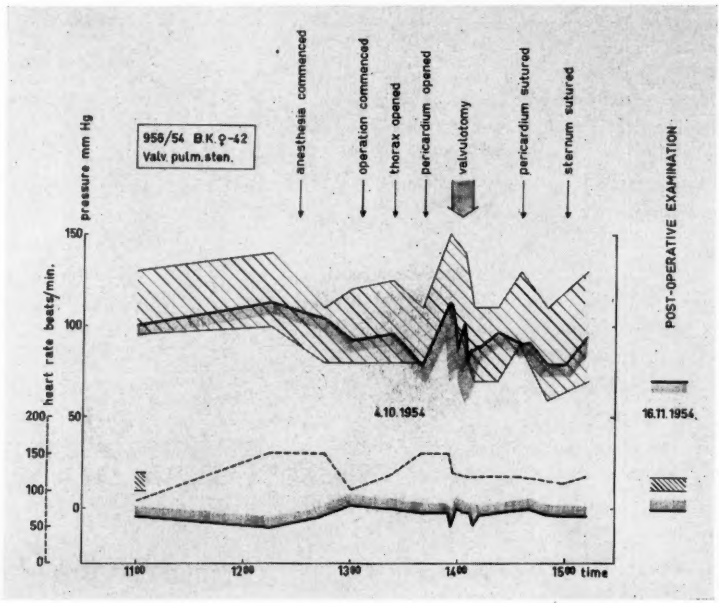

Fig. 2.

(An isolated pressure from the pulmonary artery is marked in the figure. This pressure was obtained via a catheter introduced by the operator into the pulmonary artery via the incision in the ventricular wall.)

Fig. 2 shows variations in pressure in the right ventricle in a 12-year-old girl operated upon for valvular pulmonic stenosis, ad modum Brock. The pressure values here show *lower* values *before* the valvulotomy than after the operation. As in Fig. 1, the pressure obtained on postoperative examination in the waking state is lower than any pressure recorded during the operation.

Fig. 3 illustrates the variations in pressure in the pulmonary artery during valvulotomy performed on a 4-year-old boy. A slight defect in the septum of the ventricle had been diagnosed preoperatively; after the splitting of the valve it was decided that the defect in the ventricular septum must be of considerable size, so that the effect of the valvulotomy was not favorable. During the final

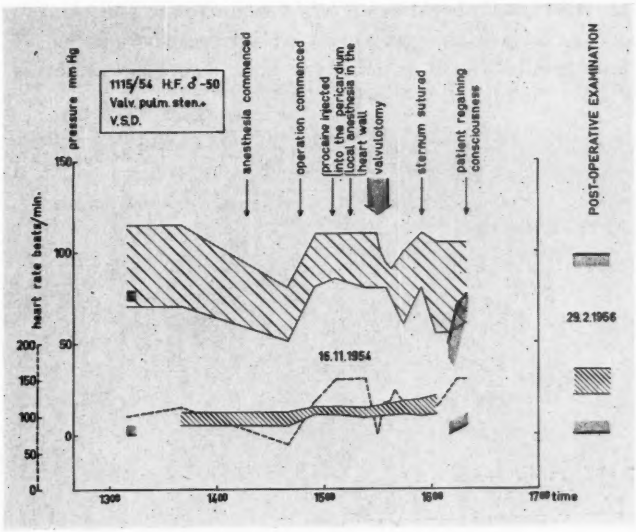
1115/54 H.F. ♂ -50
Valv. pulm. sten.+
V.S.D.
anesthesia commenced
operation commenced
procaine injected into the pericardium local anesthesia in the heart wall
valvulotomy
sternum sutured
patient regaining consciousness
POST-OPERATIVE EXAMINATION
pressure mm Hg
heart rate beats/min.
16.11.1954
29.2.1956
time

Fig. 3.

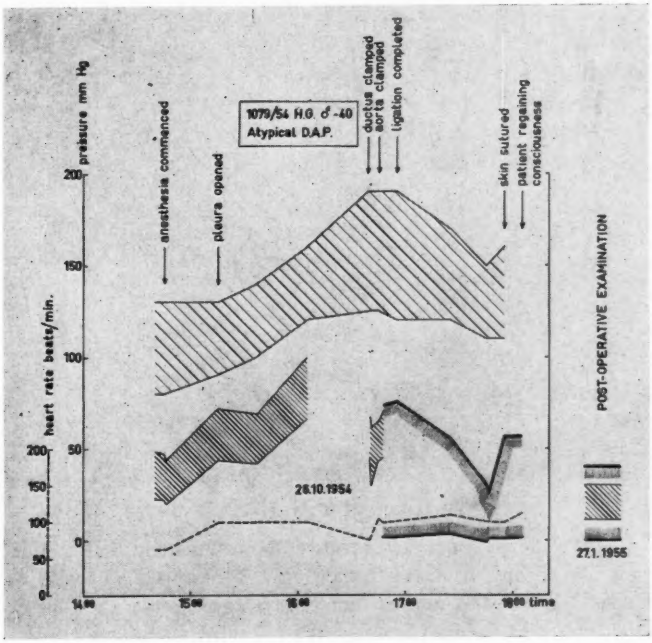
1079/54 H.G. ♂ -40
Atypical D.A.P.
anesthesia commenced
pleura opened
ductus clamped
aorta clamped
ligation completed
skin sutured
patient regaining consciousness
POST-OPERATIVE EXAMINATION
pressure mm Hg
heart rate beats/min.
28.10.1954
27.1.1955
time

Fig. 4.

stage of the operation, the catheter was withdrawn to the right ventricle, so that values from this chamber were obtained during a short period. The recordings from the postoperative examination show diverging values as compared with those from the operation itself.

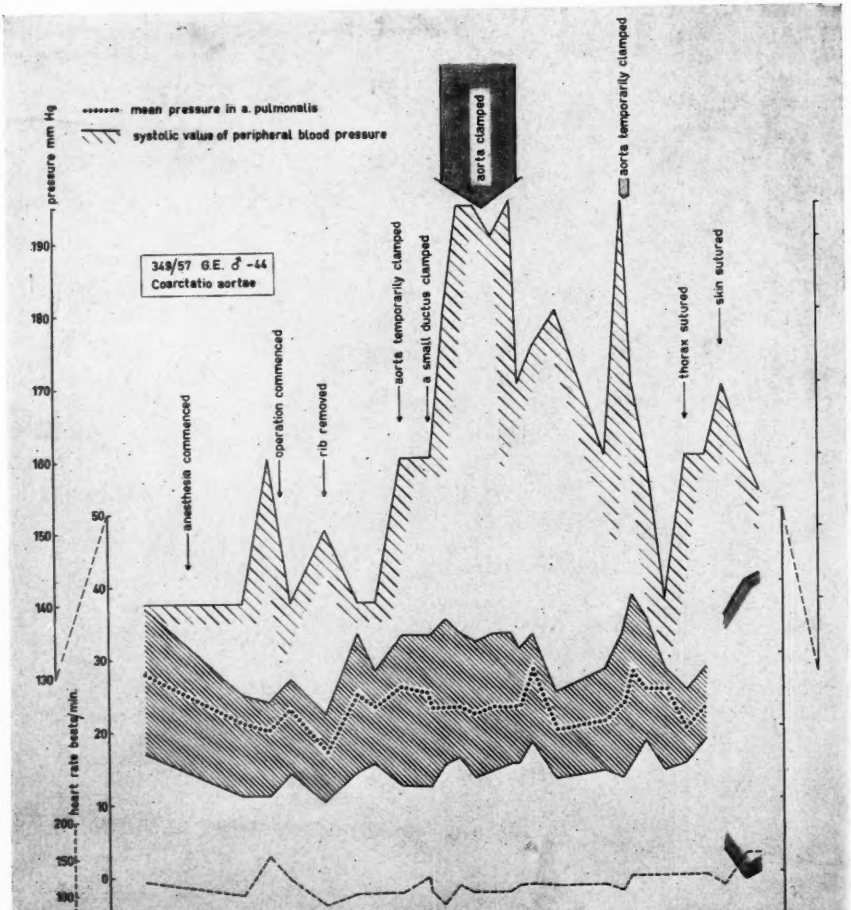

Fig. 5.

Fig. 4 shows the pressure values from the pulmonary artery and the right ventricle during an operation performed on a 14-year-old boy with an atypical patent ductus. When the ductus had been clamped, the catheter was drawn down from the right lateral branch of the pulmonary artery to the right ventricle. The pressure rose during the dissection and the manipulation of heart and vessels, fell after the clamping, appeared almost normalized about one

hour after the clamping, but rose again to show on postoperative examination 3 months later that there is still a certain hypertension in the pulmonary circulation.

(For technical reasons, intracardiac pressure recordings were not made during the period 16.05-16.40 [see Fig. 4].)

In Figs. 1-4, the right ventricle or the right ventricle and the pulmonary artery show a hypertension. By way of comparison, pressure recordings during operation were made from a pulmonary artery in which the pressures were not so high. This is shown in Fig. 5. The patient was a 13-year-old boy with coaretation of the aorta at the typical point below the branching off of the left subclavian artery.

The variations in pressure in the pulmonary circulation were moderate; a reduction was noted during the narcosis, a rise during the manipulations of heart and vessels, although not to the same extent as the changes in the peripheral pressure during the clamping of the aorta.

(As palpation of the radial pulse was applied in this case, it has only been possible to give the systolic value of the peripheral blood pressure.)

DISCUSSION

What gave rise to this investigation was the possibility of being able, by means of a continuous recording of pressures during valvulotomy, to read the effect of the operation more precisely than has been possible through isolated pressure measurements via punctures of the right ventricle wall.

The investigations presented here show that if, in these cases, isolated pressure measurements had been recorded, representative values would not have been obtained during the actual operation. Altogether, the method of isolated pressure measurement—even under relatively standardized conditions—must be regarded as a rather unsatisfactory mirror of the intracardiac hemodynamic state.[6]

By comparing the pressures recorded during operation with those recorded at a postoperative examination we have, moreover, been able to show that, in cases of pulmonary stenosis, the effect of the valvulotomy can only be judged at a control examination performed a considerable time after the operation. This has been pointed out by several writers,[7, 8, 9] but is at variance with what has been adduced in other quarters.[5] Nor are the pressure values from the pulmonary circulation (right ventricle) immediately after the closure of a large potent ductus (Fig. 4) valid at a postoperative examination 3 months later.

In operations of aortic stenosis, Brock[3] has for diagnostic purposes found withdrawal curves from the aorta during actual operation very useful. Isolated simultaneous pressures from the aorta and left ventricle immediately before and after valvulotomy are considered by Brock[3] and Bakst and his associates[1] to give a reliable notion of the result of the operation, although Brock makes the reservation that pressures during operation cannot be compared with those from a later occasion.

SUMMARY

Intracardiac pressure measurement, with the help of a catheter introduced into the heart before the beginning of the operation, is described in 3 cases of valvular pulmonary stenosis, 1 case of atypical patent ductus arteriosus, and 1 case of coarctation of the aorta. Continuous pressure recording has in this way been rendered possible during actual valvulotomy of the pulmonary valves, during clamping of a wide ductus, and in connection with clamping of the aorta during operation for coarctation of the aorta.

The advantage of this examination method as compared with the isolated pressure recordings via punctures of the heart cavity and vessels is pointed out.

The effect of valvulotomy in cases of pulmonary stenosis can only be assessed through preoperative and postoperative pressure measurements in the right ventricle, which is illustrated by comparing values from the actual operation with those obtained on postoperative examination, at least one month later.

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PRESSURE-FLOW RELATIONSHIPS OF THE SIGMAMOTOR PUMP AS USED IN CARDIAC BYPASS

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THE development and clinical application of open heart surgery utilizing extracorporeal circulation has led to wide use of the Sigmamotor† finger-type pump.[1] As an occlusive type pump, it is designed to provide a constant output despite changes in the resistance against which it is pumping. It has been assumed, then, that the pump output during cardiac bypass when the mean systemic pressure may be as high as 175 mm. Hg will be the same as the predetermined flow during calibration against zero pressure.[2] In our early experience with this pump in the laboratory, it was found that the actual output during cardiac bypass was less than the previously calibrated flow, resulting in excessive dog mortality. Subsequent studies revealed a sharp drop in pump output with increased pressure unless extreme care was taken to obtain an optimum occlusive setting of the pressure plate. The pressure-flow characteristics of this pump with and without the optimum occlusive setting and a method for obtaining the optimum occlusive setting constitute the subject of this report.

METHODS

A Sigmamotor Model T-6S cross circulation pump of a capacity of 4,500 c.c. per minute was used. The calibrations were performed using a 20 cm. long, ¾ inch I. D. gum rubber tube in the pump head, which was connected distally to a 5/16 inch I. D. plastic tube of 80 cm. in length and proximally to a similar plastic tube which included a Monel metal filter.‡

The level of the perfusing solution in the reservoir was maintained at approximately the same height as the pump head. A catheter was attached to the end of the distal limb by a tapered plastic adapter. The catheter varied in size according to the anticipated caliber of the artery to be cannulated. For these experiments the size was a No. 12 or No. 14 Fr. The occlusive setting of the pump was determined by elevating the distal plastic tube 2 or more feet above the pump head and adjusting the pressure plate to that position which

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This work was supported in part by grants from the New York Heart Association, The Hartford Foundation, and the Alfred P. Sloan Foundation.
Received for publication Nov. 20, 1957.
*Vincent Youman Memorial Research Fellow in Medicine.
**Nathan Hofheimer Research Fellow in Medicine.
†Sigmamotor, Inc., Middleport, N. Y.
‡Kindly supplied by Abbott Laboratories, North Chicago, Ill.

prevented regurgitation of the fluid through a complete cycle of the pump. When this position of the pressure plate had been determined by the maneuver described, each pressure plate screw was loosened by ¼ turn, and this setting was taken to be the optimum occlusive setting.

RESULTS

The effect of pressure on the output of the pump with a deliberately nonocclusive setting of the pressure plate for flows of from 360 c.c. per minute to 980 c.c. per minute is shown in Fig. 1. It is apparent that with an average mean systemic blood pressure of 100 mm. Hg, reductions of 60 per cent from the calibrated flow against zero pressure can occur.

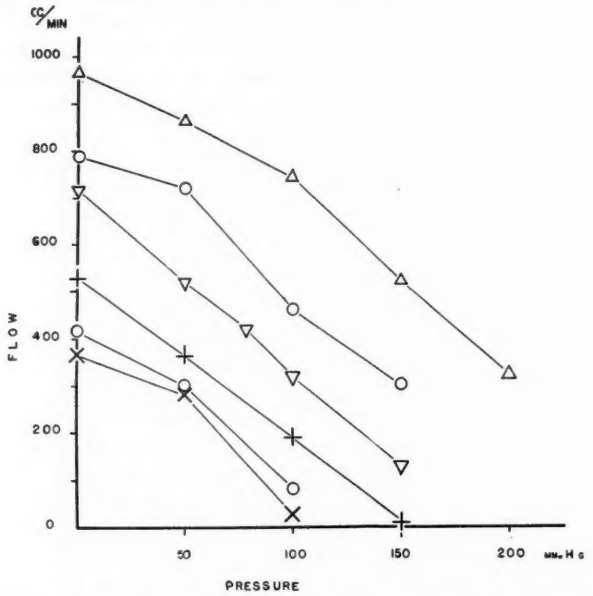

Fig. 1.—Nonocclusive Setting. The effect of pressure on pump output with a deliberately nonocclusive setting (see text for discussion.)

The effect of pressure on flow with varying degrees of nonocclusiveness as quantitated by ¼ screw turns from the optimum plate setting is shown in Fig. 2. These data indicate that minor degrees of deviation from the optimum occlusive setting can have a significant effect on pump output and that this effect is magnified by increasing pressure. With optimum occlusive setting of the pressure plate there is a negligible effect of increasing pressure up to 240 mm. Hg on flow as is demonstrated in Fig. 3.

DISCUSSION

The Sigmamotor pump, designed to operate as an occlusive pump, can be relied upon to maintain the predetermined output against increasing pressure

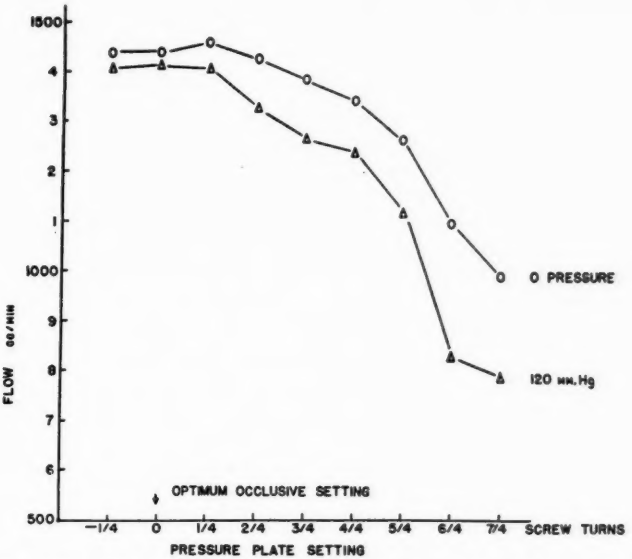

Fig. 2.—Flow With Varied Setting. The effect of peripheral pressure on pump output with varying degrees of nonocclusiveness as quantitated by ¼ turns of the pressure plate screws.

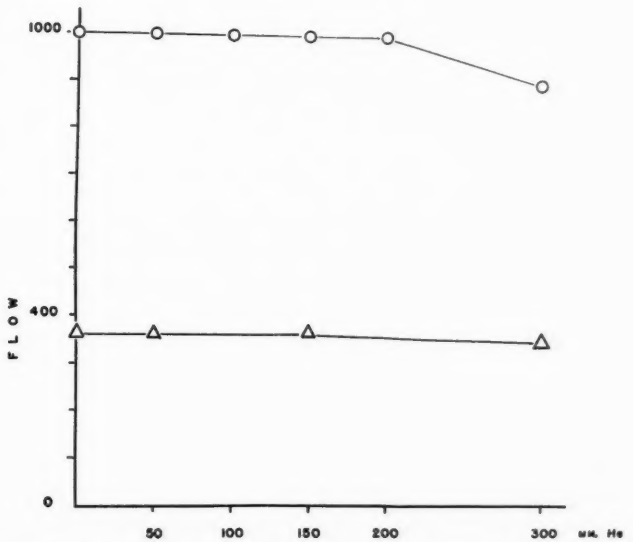

Fig. 3.—Optimum Occlusive Setting. Negligible effect of pressure on pump output with optimum occlusive setting.

only if the optimum occlusive setting of the pressure plate is verified prior to each use of the pump. Minor deviations from this optimum setting can result in significant reduction in flow when pumping against systemic pressure. In a study of the physiologic responses during cardiac bypass, mean perfusion pressures of 34 to 175 mm. Hg were reported from this laboratory.[3, 4] It is apparent, then, that during cardiac bypass when systemic pressures of this magnitude are encountered, the pump output may be reduced 5 to 60 per cent from the previously calibrated output against zero pressure if care is not taken in obtaining the correct pump setting. Such reductions in pump output may contribute to the acidosis, hypoxia, and death due to low flow, when it is thought that adequate flow is being utilized.

SUMMARY

The pressure-flow characteristics of the Sigmamotor pump with and without optimum occlusive setting of the pressure plates are presented. The necessity for obtaining the optimum occlusive setting to assure the predetermined output of the pump during cardiac bypass is emphasized. A method for verifying the optimum occlusive setting of the pressure plate is described.

ADDENDUM

Sigmamotor, Inc., has recently developed and made available a new pump head with vernier knob adjustment of the pressure plate. Thus, the need for individual adjustment of the pressure plate screws is eliminated. However, the necessity for optimum occlusive setting of the pressure plate remains of utmost importance.

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RESISTANCE TO CATHETER PUNCTURE OF VARIOUS CARDIOVASCULAR STRUCTURES

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THERE are very few untoward incidents or fatal results related to cardiac catheterization despite its wide use in the investigation of heart disease. In particular, puncture of cardiac chambers or intrathoracic vessels has rarely been reported.[1] Some canine physiologic studies were being made in this laboratory which involved the insertion of a catheter through a brachiocephalic artery. In a series of 11 dogs, 3 suddenly developed shock and eventually died. Viewed fluoroscopically, the catheter appeared to be free within the chest cavity. Thoracocentesis was performed, and pure blood was obtained. Autopsy in each instance showed a minute puncture at the origin of one of the branchiocephalic arteries from the aortic arch. These incidents aroused interest as to how much stress would puncture the great vessels of the cardiovascular system. Haddy and his co-workers[2] found a 10 per cent mortality in left heart catheterization in dogs, which included shearing of the brachiocephalic artery from the aorta, and perforation of the aorta and of the left ventricle; while none occurred with right heart catheterization. Several studies have been made on the tensile strength of strips or cylindric segments of blood vessels,[3-10] but none on resistance to impact stress on a sharply localized area of a vessel as might be made by the thrust of a cardiac catheter. A simple method of carrying out such a study was therefore devised.

MATERIALS

A Lucite platform was constructed (*A*) in Fig. 1, with a 4 mm. hole in the top plate. Another piece of Lucite (*B*) with a corresponding hole was placed over the platform, and over this hole was attached a piece of glass tubing (*C*), 2 cm. long and of 0.5 cm. internal diameter. Clamps (*D*) were provided to permit compression between the plates. Pieces (*E*) about 4 cm. long were cut off from the tips of Nos. 5 and 8 Goodale-Lubin catheters. A piece of rigid stainless steel wire (*F*), corresponding to the internal diameter of each catheter tip, was attached to a small thin piece of balsa wood (*G*). This could then be threaded through the corresponding catheter segment to provide rigidity. The wire length was adjusted so that the tip fell about 1 mm. short of the tip

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Supported by grants from the James Whitcomb Riley Memorial Association, and the Indiana Heart Foundation.
Received for publication Nov. 29, 1957.

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of the catheter. The steel wire and its balsa plate with a No. 8 catheter tip weighed about 1.2 Gm., and with a No. 5 catheter tip, 0.7 Gm. A glass tube (*H*), 30 cm. long, 2.2 cm. internal diameter, and weighing 78 Gm., with a rigid detachable bottom (a pill-bottle cap [*I*] over which a flat piece of balsa wood [*J*] was attached), was allowed to rest on the plate holding the catheter, and kept upright by loose encirclement with ring clamps. Lead weights of graduated sizes at 5 Gm. intervals, over the range from 5 to 45 Gm., were obtained.

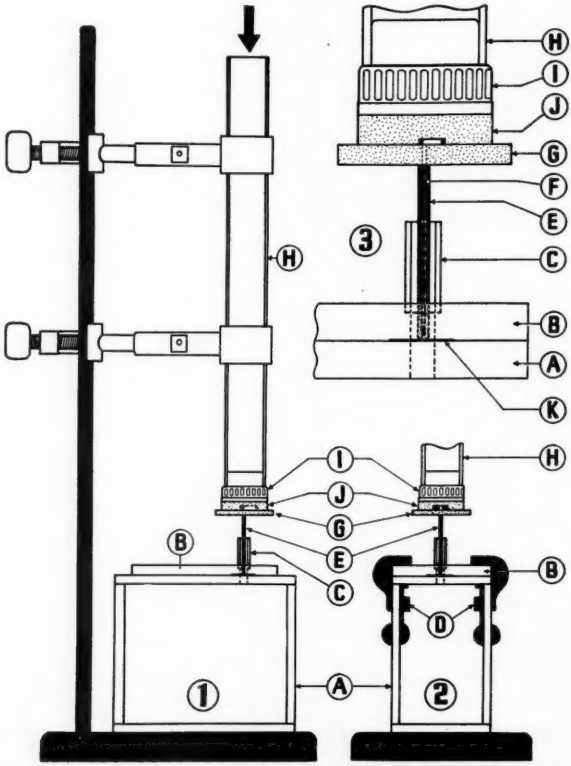

Fig. 1.—Apparatus used for testing vessel segments for relative resistance to catheter puncture. (*1*) side view; (*2*) front view, (*3*) detail of catheter tip resting on tissue. (*A*) Lucite platform, (*B*) Lucite top plate, (*C*) glass tubing, (*D*) clamps, (*E*) catheter tip, (*F*) steel wire, (*G*) balsa plate, (*H*) glass tube, (*I*) pill-bottle cap, (*J*) balsa wood attached to cap, (*K*) tissue.

The aortic arch, superior vena cava, and main pulmonary artery were sectioned from a large mongrel dog, weighing 31 pounds. Aortic arches were also obtained from 3 other dogs of various sizes.

METHOD

A segment of blood vessel was carefully split open and placed across the hole on the Lucite platform. The other piece of Lucite was laid across the

platform, and the 2 plates were clamped together to hold the tissue in place (*K* in Fig. 1), without placing it on stretch. The catheter tip assembly was placed through glass tube (*C*) with its end supported by the tissue. The glass tube (*H*) was placed on the balsa plate of the catheter tip and allowed to rest freely. Lead weights were carefully dropped into the tube from a fixed point at the top, starting with the 5 Gm. weight, and progressing to the next higher weight until the tissue was punctured. Each weight was removed from the tube before dropping the next one. The piece of glass tubing attached to the upper piece of Lucite supported the catheter tip so that it did not bend when a weight was dropped. The vessel was therefore subjected to a fixed static load of the combined weights of the glass tube and catheter set-up resting on the tissue (approximately 80 Gm.), plus the impact load of the graduated weights dropped through a fixed distance. Pilot experiments showed that additional static loading of approximately 1,600 Gm. or 20 times the static loads applied in the apparatus were necessary to produce rupture of these vessels held in this manner. Thus, the static loads used in the experiment were of minor significance in comparison to the impact loads. With distance held constant, the weights necessary to produce rupture on impact should constitute reliable indices of relative resistance to puncture.

The superior vena cava, main pulmonary artery, and aortic arch from one dog were tested. Determinations were also made at the origin of the brachiocephalic arteries from the aortic arch from this dog, as well as from 3 other dogs. All tissues were kept wet with normal saline solution throughout the experiment.

RESULTS

The results of 30 determinations on each great vessel of 1 dog, and also on a number of points along the aortic arches of 3 other dogs are shown in Tables I, II, and III.

DISCUSSION

Very few animals were used, for it was not the purpose to establish absolute values in dogs, but rather to compare tissue strength. Furthermore, it was expected that the possible effects on tissue structure of factors such as age, size, diet, and habits of the animal would exert the same influence on each determination of tissue strength. The significance of mean differences in puncturing weights of the different vessels were tested for by computing t ratios.[11]

As indicated in Table I, the difference in strength of the superior vena cava and the aorta was small. Nevertheless, the difference was sufficient to have occurred only 5 to 10 per cent of the time on the basis of chance. The results, then, suggest a possibility that the resistance to puncture of the superior vena cava is slightly greater than that of the aorta. The aorta and the superior vena cava were over 1.5 times as strong as the main pulmonary artery, a difference shown at a very high level of significance. It took a greater amount of weight to puncture the aortic arch with a No. 8 catheter than with a No. 5 catheter as might be expected with the force being resisted by a larger area of vessel in the case of the No. 8 catheter. There was a significant weakness at

the points where the brachiocephalic arteries originated from the aortic arch, especially at the proximal curve (Tables II and III). Pathologic changes have been found to occur more where the branches leave the aorta.[12] Since the most rapid flow occurs along the shortest course at a ramification, the proximal angle of a branching is exposed to more mechanical trauma than the distal angle. In this study the proximal curve of the right brachiocephalic artery as it came off the aortic arch was compared with the area between the two brachiocephalic arteries. The distal curve of the left brachiocephalic artery was not evaluated for comparative study because of the fewer determinations made at this point.

TABLE I*

	SUPERIOR VENA CAVA	MAIN PULMONARY ARTERY	AORTIC ARCH	AORTIC ARCH (NO. 8 CATHETER)
Mean	17.66	10.50	16.33	23.83
S.D.	2.22	1.35	2.28	2.76
Range	15-20	10-15	10-20	20-30
N	30	30	30	30

TABLE II*

	ORIGIN OF BRACHIOCEPHALIC ARTERIES				
	PROXIMAL	DISTAL	BETWEEN BRANCHES	TOTAL	AORTIC ARCH
Mean	11.36	14.28	14.58	13.33	15.33
S.D.	2.16		2.32	2.67	1.59
Range	10-15	10-20	10-20	10-20	10-20
N	11	7	12	30	30

TABLE III*

TISSUES UNDER COMPARISON	DIFFERENCE OF MEANS	t	p
Superior vena cava and main pulmonary artery	7.16	13.04	$<.001$
Superior vena cava and aortic arch	1.33	1.97	$<.10; >.05$
Main pulmonary artery and aortic arch	5.83	10.50	$<.001$
Aortic arch with Nos. 5 and 8 catheters	7.50	10.00	$<.001$
Total points of origin of branch arteries and aortic arch	2.00	3.08	$<.01$
Origin of branches: at proximal curve and between branches	3.22	3.16	$<.01$

*All weights are expressed in grams and refer to weight dropped through a constant distance in the described apparatus which produced rupture of the tissue. N = number of determinations. S.D. = standard deviation.

All determinations were made with a No. 5 catheter unless otherwise stated.

SUMMARY

1. A simple method was devised for testing comparative tissue resistance to catheter puncture stress in the main pulmonary artery, superior vena cava, aortic arch, and points of origin of the brachiocephalic arteries in dogs.

2. The results suggested slightly increased tissue resistance in the superior vena cava as compared with the aortic arch. The main pulmonary artery was weaker compared with these two vessels.

3. There was a significant weakness at the points where the brachiocephalic arteries left the aortic arch, especially at the proximal curve, compared with the aortic arch.

4. More stress was needed to puncture the aortic arch with a No. 8 catheter than with a No. 5 catheter.

We gratefully acknowledge the expert statistical advice of Dr. Ralph Reitan, Associate Professor of Psychology, Indiana University School of Medicine.

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DIRECT VISION APPROACH TO THE AORTIC VALVE WITHOUT OCCLUSION OR BYPASS

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INTRODUCTION

A TECHNIQUE is presented which permits exposure of an aortic cusp through the sinus of Valsalva without circulatory occlusion or bypass. A clamp (Fig. 1)† consisting of two coapting baffles, one fenestrated and the other recessed, is positioned over the noncoronary aortic cusp to form a chamber in which direct visualization and manipulation of the cusp are possible. Tailoring of all or part of the cusp and replacement by a graft or prosthesis have been carried out on experimental animals. The operating time within the valve ring is relatively unlimited since neither extracorporeal circulation nor vascular occlusion is required.

PROCEDURE

One hundred and three adult mongrel dogs, weighing 12 to 28 Kg., have been operated on. Intravenous Nembutal anesthesia is administered, an endotracheal tube inserted, and ventilation maintained with an Emerson, pressure-sensitive, cycling respirator. The inspiratory pressure varies from +14 to +18 cm. H_2O and the expiratory pressure from +4 to +6 cm. H_2O. After positioning the dog for a right thoracotomy (Fig. 2), a control electrocardiogram is taken and the cardiogram is monitored during the procedure with a Sanborn oscilloscope. A small midline incision is made and the xyphoid is resected in the animals having autogenous graft valvular replacement. The xyphoid is tailored to form a cartilaginous frame. This incision is closed and a standard intercostal incision is made in the right third interspace. A modified Finochietto retractor is inserted. The right lung is retracted caudally to expose the base of the heart. A 3 by 4 cm. segment of pericardium (Fig. 3) is removed, defatted, and the free valvular graft is constructed. A pericardial envelope is made, inverted to place the suture line inside, and the tailored xyphoid stent is inserted. The flap is immersed in dilute heparin solution until needed. Following re-expansion of the lungs, the right lung is again retracted caudally with a moist sponge. A traction suture in the tip of the right atrial appendage is used to retract the appendage, exposing the base of the aorta (Fig. 4). Control aortic and left ventricular pressure pulses are then

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This work was supported in part by a grant from The Plymouth Chapter of the Massachusetts Heart Association.

Received for publication Dec. 13, 1957.

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†The baffle clamp was constructed by Codman and Shurtleff, Inc., 104 Brookline Ave., Boston, Mass.

recorded. The periaortic fat is excised along the posterior and lateral surfaces of the ascending aorta. Care must be taken to avoid the right coronary artery at its origin. The posterior sinus of Valsalva can be easily palpated distal to the junction of right atrium and aorta on the lateral side of the outflow tract. A partial occlusion clamp is applied to the lateral wall of the ascending aorta approximately 3 cm. distal to the sinus of Valsalva. A 1.5 cm. longitudinal

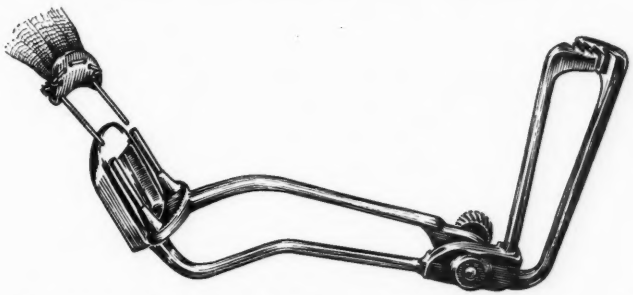


Fig. 1.—Drawing of locked baffle clamp. Sliding retractor of fenestrated baffle can be removed when pedicled graft is used.

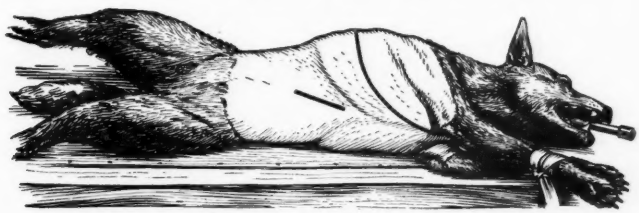


Fig. 2.—Xyphoid and third right intercostal space incisions.

incision is made in the aorta (Fig. 5), and a pericardial or plastic pouch is sutured to the edges of the incision[1, 4] (Fig. 6). The pouch is filled with dilute heparin solution. After insertion of the internal recessed baffle of the baffle clamp (Fig. 7), the purse-string suture of the pouch is tied and reinforced. The partial occlusion clamp is gradually released. Sponge pressure helps to attain hemostasis when necessary. The internal baffle is advanced into the aortic ring (Fig. 8) and the fenestrated external baffle is then positioned over the posterior sinus of Valsalva. The clamp is locked, forming a chamber which contains the posterior sinus of Valsalva with its aortic cusp. The wall of the aortic sinus is incised transversely for a distance of 1.0 to 1.2 cm. to expose the underlying leaflet (Fig. 9). Cardiac action throughout these maneuvers is not markedly altered since circulation is not occluded. The lunular edge of the cusp is grasped and the leaflet may be partially or completely excised.

If a graft is to be inserted, mattress sutures of 4-0 silk are placed through the vessel edges and graft base before advancing the graft into the chamber (Fig. 10). This obviates the possibility of inadvertently immobilizing the flap with a poorly placed suture. When the graft is in correct position, the mattress

sutures are loosely tied. A second layer of mattressed adventitial sutures is then taken. Pressure is applied to the suture line for 10 minutes as the baffle clamp is unlocked. The unlocked clamp is dismantled, and the recessed baffle is returned to the pouch, permitting the graft to flap between the remaining leaflets and the aortic wall (Fig. 11). The pouch is excluded by replacing the partial occlusion clamp to permit its removal. The distal aortic incision is closed with mattress sutures and oversewn. Pressure measurements are again recorded.

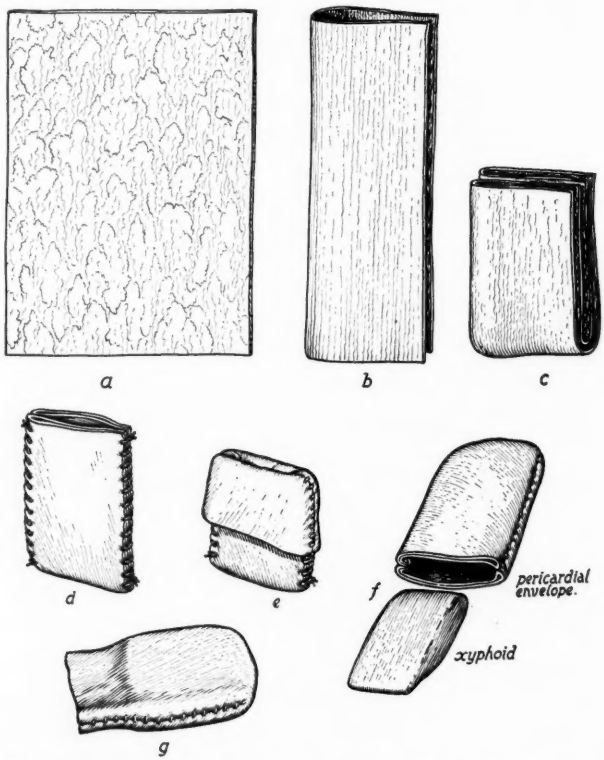


Fig. 3.—Preparation of free autogenous flap from pericardium and xyphoid. Pericardial envelope is inverted to place suture line inside.

The pericardium is ordinarily not approximated. The chest is closed over an intercostal tube connected to an underwater seal. The lungs are expanded and the intercostal tube is removed. Spontaneous respirations return within seconds of disconnecting the respirator. The incisions are coated with collodion, another electrocardiogram is recorded, and the endotracheal tube is removed.

Postoperatively, the dogs receive intramuscular penicillin for 7 days. There is no restriction of activity postoperatively and the dogs are not digitalized.

Pressure measurements are made with Statham strain gauges (P23A-P23D) connected to a Sanborn 150 M 4-channel recorder monitored with a Sanborn Visoscope. The manometers are all balanced to the same fluid level arbitrarily set at table-top height.[3, 7]

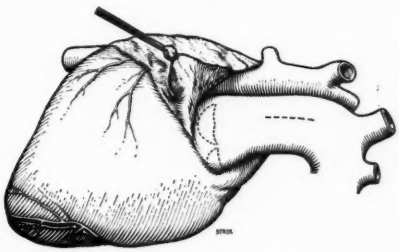


Fig. 4

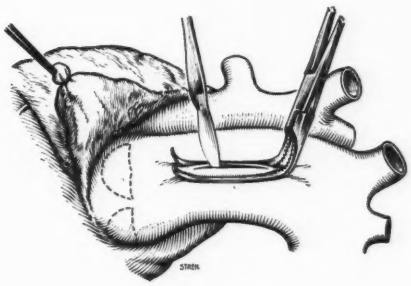


Fig. 5

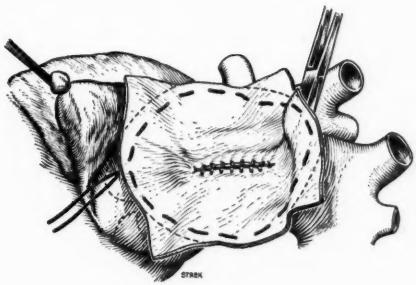


Fig. 6

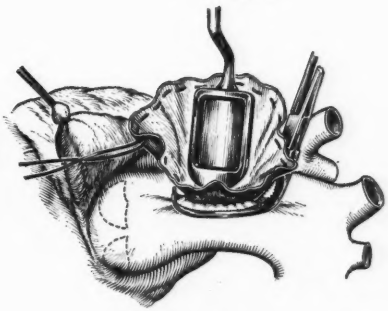


Fig. 7

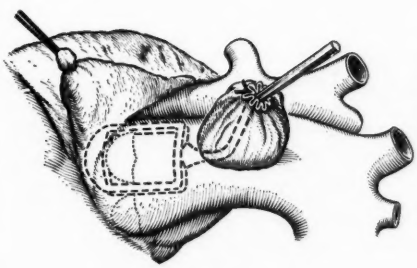


Fig. 8

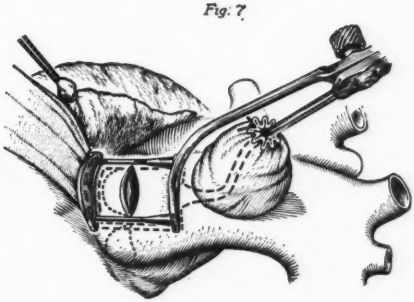


Fig. 9

Fig. 4.—Posterior sinus of Valsalva exposed by traction on right atrial appendage. Line of distal aortic incision is shown.

Fig. 5.—Partial occlusion clamp applied to aorta and distal incision made.

Fig. 6.—Pericardial or plastic envelope sutured to edges of incision.

Fig. 7.—Recessed baffle inserted into pouch.

Fig. 8.—Purse-string suture tied. Recessed baffle advanced to region of posterior sinus of Valsalva.

Fig. 9.—Fenestrated baffle applied, positioned over aortic sinus, and clamp locked in place. Transverse incision made over lunular edge of posterior (noncoronary) aortic cusp.

Fig. 10.

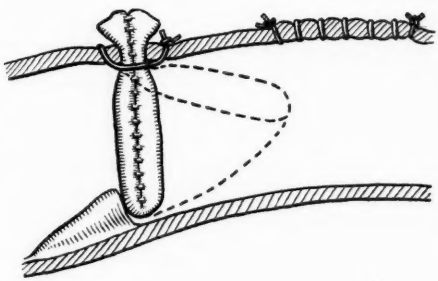


Fig. 11.

Fig. 10.—Sutures placed in aorta and free graft before advancing graft into chamber.
Fig. 11.—Graft swings free with systole and engages remaining cusps during diastole.

TECHNICAL VARIATIONS

The experiment has been set up to permit evaluation of the regurgitation produced and the effectiveness of the corrective measures. A control series of animals has had cusp resection only. Another group has had cusp resection followed by direct vision replacement with a graft or prosthesis. To date, our primary interest has been cusp replacement with an autogenous tissue graft. Pedicled pericardial grafts with cartilaginous stents theoretically seemed to offer greater promise of survival because of the retained blood supply. The theoretical advantage was decidedly outweighed by the greater technical ease with which the free grafts could be handled. There has not yet appeared to be any significant benefit from maintaining the blood supply. Woven nylon fabric has been used both to buttress the suture line and to replace the pericardium. The tendency of the fabric to fray under tension, even when the edges have been heat fused, has limited its usefulness.

The problem of operative hemorrhage prompted us to use both hypothermic and hypotensive anesthesia to minimize bleeding. Hypothermia, in this limited experience, did not cause significantly great falls in blood pressure at the therapeutic range (28° to 31° C.) to be of help. Considerable decrease in aortic tone and hemorrhage was obtained by using a hypotensive drug (Thiophanium). The dogs were extremely sensitive to the drug, however, and total doses of as little as 0.5 mg. produced profound and irreversible hypotension in 3 of 8 large dogs. Fractional injection and continuous intravenous drip of extremely dilute solutions were tried but the results were unpredictable. Reversal of the precipitous hypotensive effects by peripheral vasoconstrictors did not materially alter survival.

Although most of the animals were anesthetized with intravenous Nembutal (25 mg./Kg.), Vinethene-ether anesthesia was also employed.

On occasions, when manipulation of the right atrium produced arrhythmias, procaine was infiltrated into the sino-atrial node. These dogs were

normothermic. Bradycardia followed the production of a satisfactory S-A nodal block, but the subsequent response of the animals to even moderate hemorrhage was catastrophic. We could not help but feel that blockade of the sino-atrial node in normothermic animals served to rob them of an effective defense mechanism against shock.

RESULTS AND COMPLICATIONS

Survivals have been followed for periods up to 16 months. The mortality statistics cannot be evaluated as a group since several technical modifications have been made. The last 10 dogs were operated on by the technique described with one operative fatality (10 per cent). This dog had electrocardiographic evidence of an anterior myocardial infarction. The oscilloscopic tracing revealed S-T segment displacement as the posterior cusp was being resected. This rapidly progressed to ventricular fibrillation and death. The animal was autopsied with the baffle clamp still locked in place. There was no encroachment on the coronary ostia by the clamp, and dissection of the coronary arteries and myocardium was uninformative. The dog had had moderately profuse hemorrhage from the pericardial pouch with no blood replacement.

In contrast to this group, there were 10 operative deaths in the first 20 dogs operated on by a similar but less refined technique (50 per cent). Hemorrhage was the precipitating factor in 7 of these. Four of the 7 hemorrhagic deaths were due to faulty construction of the baffle clamp. There was tearing of the aorta from sharp angles present on the original clamp. Utilization of an inadequate aortic pouch caused 2 deaths in this group, and unsatisfactory hemostasis of the aortic sinus suture line led to the demise of the third. Care is now taken to avoid even slight tension of the proximal aortic incision to prevent intimal tears when the baffle clamp is released. One dog developed ventricular tachycardia following cardiac manipulation. Pronestyl was administered but a 3:1 A-V block supervened. This progressed to fibrillation which could not be reverted electrically. Another dog died following inadvertent transfusion of blood containing 75 ml. of sodium citrate solution after closure of the chest. One dog had marked electrocardiographic changes at the completion of the procedure suggesting an acute myocardial infarction. No gross or microscopic pathologic changes were found.

Several complications of the entire group of animals warrant consideration. Biochemical analyses during the earlier procedures revealed acidosis in many of the dogs with blood pH ranging to 7.13. This was shown to be respiratory acidosis precipitated by a subatmospheric expiratory phase of the respirator. Adjustment of the respirator to maintain positive pressure cycling from +16 on H_2O to +4 H_2O at 20 to 30 excursions per minute has corrected this abnormality.

There have been 5 late postoperative deaths in the 103 animals in which bronchopleural fistula was either a major or contributing factor. The fistulas originated in pulmonary parenchymal tears sustained at operation. Five dogs died from empyema. Embolization of the flap valve occurred in 2 animals who

died 8 days and 2½ months following surgery. One operative fatality resulted from embolization to the brain and aortic trifurcation of a thrombus formed in the aortic pouch.

Pulsus alternans has been noted during operation in approximately 5 per cent of the procedures. The etiology has not been determined, but it has had no prognostic significance. This is in sharp contrast to the gravity of pulsus alternans clinically.

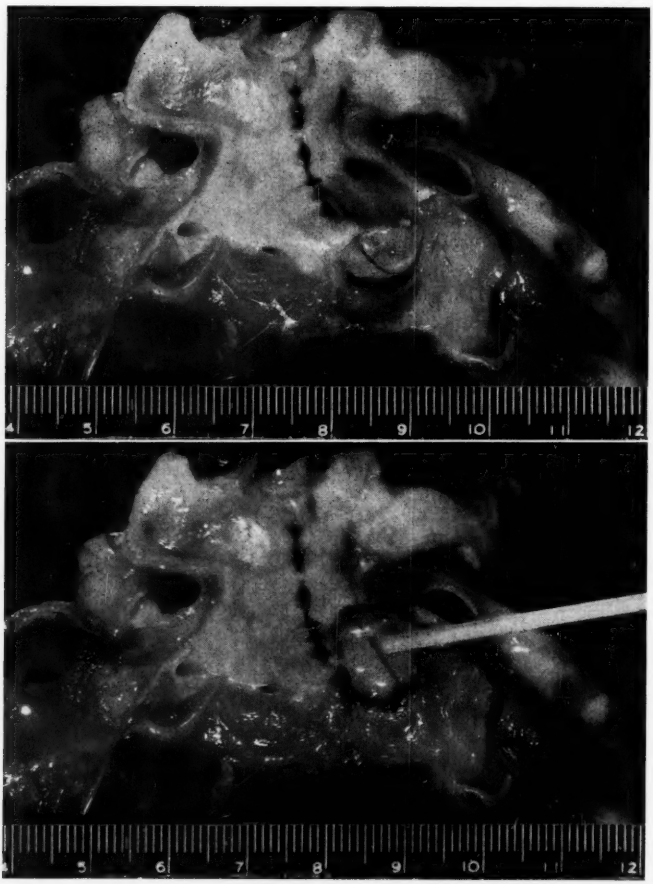
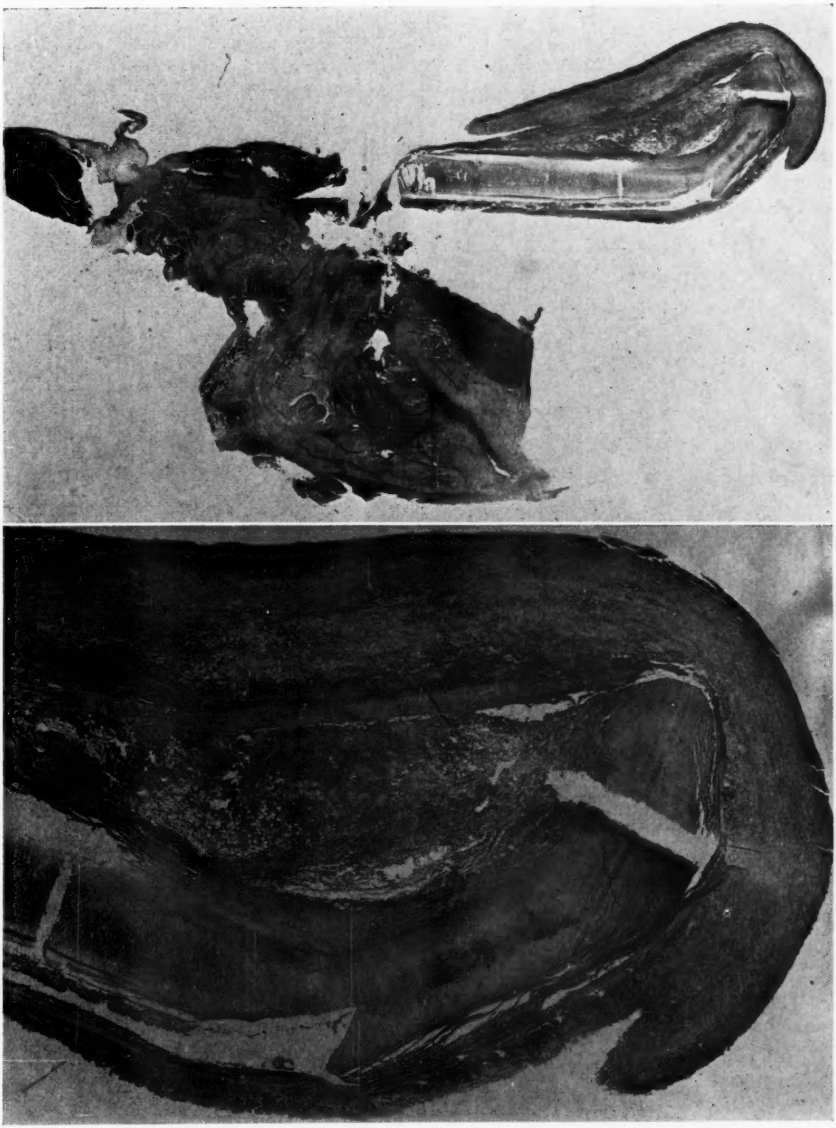


Fig. 12.—Appearance of free graft in animal sacrificed 11 weeks after excision of posterior cusp with autograft replacement. *A*, Position during diastole. *B*, Position during systole.

The fate of the autogenous flap valves is being studied. The animals have been followed for periods up to 16 months and are being sacrificed at calculated intervals (Figs. 12, 13). The principal postoperative difficulty encountered has been suture-line hemorrhage precipitated by intimal tears at the

A.



B.

Fig. 13.—*A*, Low-power photomicrograph of graft in animal sacrificed 3 weeks postoperatively. Organized thrombus present on proximal side.
B, Higher power photomicrograph of graft.

proximal edge of the aortic sinus incision. This problem appears to have been obviated by eliminating suture-line tension. Prosthetic valves other than nylon-covered stents have not yet been evaluated.

Intracardiac operating times have varied from 30 minutes to 180 minutes with survival. The presence of the clamp is generally well tolerated if care is taken to avoid angulation of the ascending aorta. We have been able to demonstrate circumscribed areas of pressure necrosis from the clamp in only 2 animals. In each, incorrect application of the clamp on the base of the right atrium produced distortion.

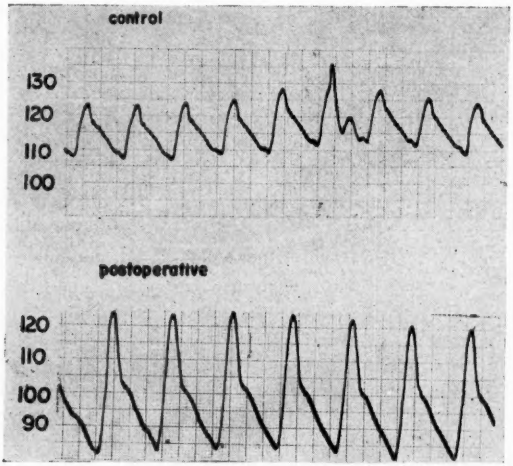


Fig. 14.—Aortic insufficiency without cusp replacement. Aortic pressure tracings before and after production of valvular regurgitation by partial excision of posterior cusp.

HEMODYNAMIC OBSERVATIONS

Uncorrected aortic insufficiency was produced in 9 animals by partial or complete excision of the posterior (noncoronary) aortic leaflet (Fig. 14). Pulse pressures were increased an average of 4 times, the smallest increase changing from 25 mm. Hg to 40 mm. Hg and the largest deviation from 15 mm. Hg to 85 mm. Hg (Table I). The dogs were permitted moderate activity and were not digitalized. The longest survival was 10½ weeks. There was one operative fatality, one animal died from cardiac tamponade 2 weeks after surgery, and 7 dogs succumbed from pulmonary congestion and edema. At autopsy the 7 dogs manifested no evidence of cardiac dilatation or increased pericardial tension.

A completed series of dogs in whom cusp excision was followed by valvular replacement (Fig. 15) is not yet available since an acceptable mortality rate for this combined procedure has only recently been attained. The last 7 dogs in whom this technique was carried out can be compared with the 7 dogs in whom uncorrected aortic insufficiency was produced and pressure measurements obtained (Table II). No conclusions can be drawn from this series since

no attempt was made to restore blood pressures to preoperative levels (Fig. 16). The animal in whom a twofold variation occurred (Feb. 27, 1957) died on March 7, 1957. Autopsy revealed that the flap had embolized leaving pure aortic insufficiency. The cause of death was pulmonary congestion and edema.

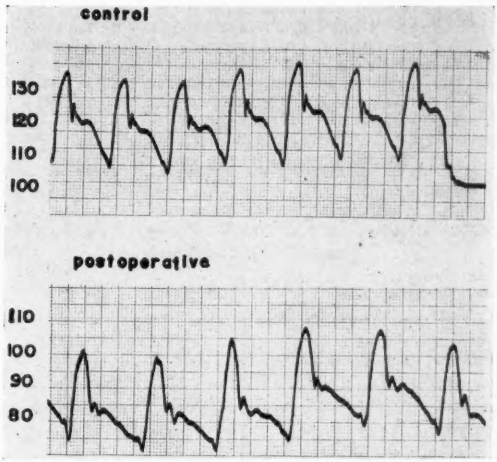


Fig. 15.—Aortic insufficiency with cusp replacement. Aortic pressure tracings before valvular surgery and after production of aortic insufficiency with autogenous cusp replacement.

TABLE I. EXPERIMENTAL AORTIC INSUFFICIENCY*

DATE	PROCEDURE	PREOPERATIVE AORTIC PULSE PRESSURE (MM. Hg)	POSTOPERATIVE AORTIC PULSE PRESSURE (MM. Hg)	FATE
1. Dec. 27, 1956	Cusp resected	Aorta angulated inadvertently		Operative death
2. Jan. 3, 1957	Cusp resected	Pressures not obtained		Died, Jan. 7, 1957; pulmonary edema and congestion
3. Jan. 7, 1957	Cusp resected	15	85	Died, Jan. 18, 1957; pulmonary edema and congestion
4. Jan. 8, 1957	Cusp resected	25	40	Died, March 21, 1957; pulmonary edema and congestion
5. Jan. 10, 1957	Cusp resected	15	40	Died, Jan. 10, 1957; pulmonary edema
6. Jan. 11, 1957	Cusp resected	13	55	Died, Jan. 25, 1957; cardiac tamponade
7. Jan. 16, 1957	Cusp resected	10	43	Died, Feb. 3, 1957; pulmonary edema and congestion
8. Jan. 18, 1957	Cusp resected	13	60	Died, Jan. 27, 1957; pulmonary edema and congestion
9. Jan. 23, 1957	Cusp resected	7	28	Died, Feb. 4, 1957; pulmonary edema and congestion
	Mean Pulse Pressure	14	50	

*Alterations of pulse pressure produced by resection of the posterior (noncoronary) aortic cusp.

TABLE II. EXPERIMENTAL AORTIC INSUFFICIENCY WITH INSERTION OF FREE AUTOGENOUS FLAP VALVE*

DATE	PROCEDURE	PREOPERATIVE AORTIC PULSE PRESSURE (MM. Hg)	POSTOPERATIVE AORTIC PULSE PRESSURE (MM. Hg)
1. Jan. 29, 1957	Cusp resected and graft inserted	20	33
2. Feb. 11, 1957	Cusp resected and graft inserted	22	35
3. Feb. 19, 1957	Cusp resected and graft inserted	23	31
4. Feb. 27, 1957	Cusp resected and graft inserted	15	33
5. Mar. 5, 1957	Cusp resected and graft inserted	18	16
6. Apr. 8, 1957	Cusp resected and graft inserted	25	35
7. Apr. 25, 1957	Cusp resected and graft inserted	25	30
	Mean Pulse Pressure	21	30

*Pulse pressure alterations of last 7 dogs having cusp resection and replacement with free autogenous flap valve.

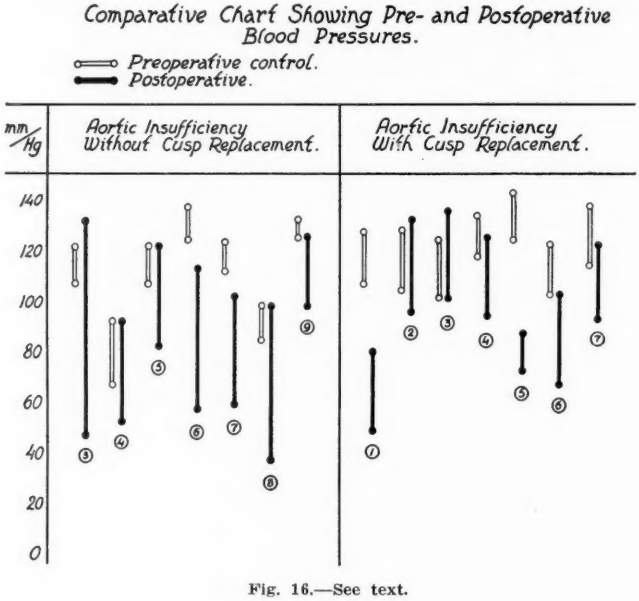


Fig. 16.—See text.

DISCUSSION

Naturally occurring aortic stenosis and aortic regurgitation are generally well tolerated for long periods of time, but both are serious lesions. It has been shown experimentally and clinically that acutely produced insufficiency is handled poorly since compensatory mechanisms cannot develop. The adherent but noncalcified stenotic aortic valve responds well to dilatation and separation of the commissures. The pathologic changes in a rigid, calcified valve, though, do not readily succumb to such treatment. Removal of a portion (⅓ or more) of the deformed valve restores normal hemodynamic relationships[2] but is not clinically feasible because of the more severe regurgitant lesion produced. Therefore, total successful restoration of the diseased aortic valve depends on a satisfactory method for controlling regurgitation,

To date the only clinically acceptable technique for the treatment of aortic insufficiency has been the Hufnagel valve.[5] Any device inserted distal to the aortic valve ring, however, reduces the regurgitant work load at the expense of the diastolic phase of coronary arterial filling. It also requires an additional procedure if direct attack on the aortic valve is desired.

The ideal approach to the aortic valve, permitting direct vision for a prolonged time with insignificant mortality, has not been achieved. A bloodless chamber inserted without interruption of the circulation has given limited access to the aortic valve without limiting operating time. Exposure of the valve ring, moreover, permits placement of a graft or prosthesis at a level which will not affect coronary arterial flow. With the development of a satisfactorily durable graft or prosthesis, it should be possible to excise a central portion of the valve and utilize the remaining rim of calcific tissue to seat the corrective device.

SUMMARY

1. An experimental technique is described which permits direct visual approach to the aortic valve without circulatory occlusion or bypass. One hundred and three dogs have been operated upon.

2. The early operative mortality was 50 per cent. The present operative mortality is 10 per cent.

3. Intracardiac operating times with survival have varied from 30 to 180 minutes.

4. Pure aortic regurgitation has been produced by resection of all or part of the posterior (noncoronary) aortic cusp. No dog survived more than 10½ weeks.

5. Aortic regurgitation has been produced in a similar group of animals and simultaneously treated by insertion of a flap valve at the aortic ring. Survivals have been followed for periods up to 16 months.

6. The fate of autogenous flap valves and prostheses is being studied.

We wish to thank Dr. John J. Byrne, director of the Third (B.U.) Surgical Service and Research Laboratory, for his continued encouragement, Dr. James W. Dow and Dr. Eric Berglund for physiologic counselling, Mr. Joseph Ferrara and Misses Joan Hartigan and Paulette Burstein for their technical assistance, and Miss Violet Peters for the preparation of the manuscript.

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PNEUMONECTOMY FOR LEIOMYOSARCOMA

Patient Well 21 Years Later

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New Orleans, La.

PNEUMONECTOMY for pulmonary disease is now so widely used that it is difficult to realize that the first successful resection was not accomplished until April, 1933.[3] The great strides that have been made in thoracic surgery since then have occurred during the experience of many active surgeons. The first patient on whom one of us (A. O.) performed a pneumonectomy was seen recently, on the twenty-first anniversary of his operation. The rarity of pulmonary leiomyosarcomas and the long survival after pneumonectomy prompted us to report this case.

REPORT OF CASE

A white man, aged 19 years, was admitted to Touro Infirmary, March 10, 1936, complaining of spitting blood and pain in the left side of the chest. In the spring of 1935, he had consulted a physician because of cough and thoracic discomfort, but no abnormalities were discovered. By July, 1935, weakness and shortness of breath had developed. By October the cough had become productive and the sputum contained streaks of blood. On Nov. 12, 1935, the patient was examined at Sanatorium, Mississippi, and told that he had pleurisy, which could be tuberculous, although no tubercle bacilli were detected in the sputum. During late December, he had a severe attack of hemoptysis and spit blood for 3 days.

On Jan. 24, 1936, he returned to Sanatorium, Mississippi, for another examination, which showed extensive atelectasis of the left lung. Dyspnea gradually increased and episodes of hemoptysis recurred. The patient had constant pain in the chest, became anorexic, and lost 10 pounds in weight.

When admitted to Touro Infirmary, the patient was pallid and undernourished. The temperature was 99.6° F. and respirations, 20. Absent breath sounds and dullness to percussion on the left together with complete opacity of the left hemithorax and shift of the heart and mediastinum to the left, demonstrated in roentgenograms, indicated complete atelectasis of the left lung.

On April 2, 1936, a soft vascular tumor completely occluding the left main-stem bronchus was seen bronchoscopically. The tumor bled freely when tissue was obtained for biopsy. The histologic interpretation of this tissue was fibrosarcoma or leiomyosarcoma. On April 13, 1936, artificial pneumothorax was induced and the left pleural cavity was half filled with air.

Left pneumonectomy was performed on April 15, 1936. About 500 c.c. of serous fluid was found in the left pleural cavity. The left lung was firm and airless, and many nodes were palpable at the left hilum. Vessels at the hilum were individually ligated. The bronchus was doubly clamped and divided. The pathologist who examined the lung immediately after its removal reported that there was no bronchial tumor within the specimen. The bronchoscopist, who was present at the operation, stated that the tumor was just beyond the tracheal bifurcation. Thereupon the remaining segment of the left main bronchus was excised completely, including a portion of the trachea, which was closed obliquely. In this removed portion of the bronchus there was a tumor which largely filled the bronchus.

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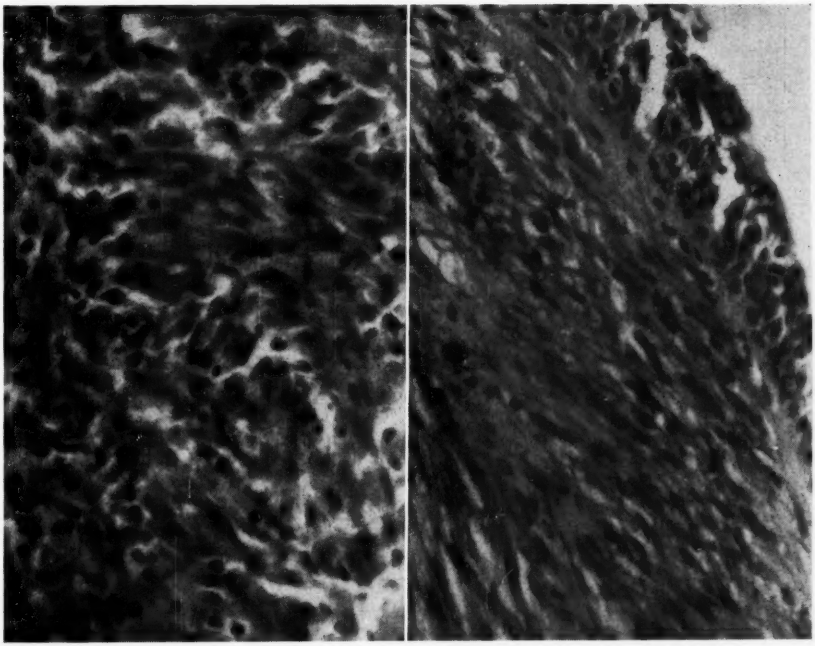
Received for publication Oct. 28, 1957.

The neoplasm was soft and pale red with scattered, dark bluish-black spots. The histologic diagnosis was leiomyosarcoma, of low-grade malignancy (Fig. 1). Only inflammatory changes were found in the enlarged lymph nodes.

Postoperatively, empyema developed in the left pleural cavity and required open drainage on April 28, 1936. The patient gradually improved after this and was discharged from the hospital on June 16, 1936.

During the next 15 years the patient considered himself well, but noticed an occasional discharge of suture material from the thoracic incision. When seen in 1953, he was working as an operator of a bulldozer. Localized empyema had developed with a long sinus tract which did not show any tracheobronchial communication. This sinus tract was unroofed and a Schede type of thoracoplasty was performed. Recovery was prompt and he returned to full time work. He has been seen intermittently since then. On April 5, 1957, he was well and working.



A. *B.*

Fig. 1.—*A* and *B*, Photomicrographs of two regions of the leiomyosarcoma arising from the bronchus. (×430; reduced ⅝.)

DISCUSSION

Before the first successful pneumonectomy by Graham,[3] many surgeons were devising surgical techniques, performing pneumonectomies on experimental animals, and attempting resection of greater and greater portions of the lung. As early as 1920, Sauerbruch[9] had accomplished a lobectomy and, in 1931, Churchill[2] had resected two lobes of the right lung. In 1931, Nissen[6] had ligated the hilar structures of a bronchiectatic lung and it had subsequently sloughed out; a similar case was reported in 1933 by Haight.[4]

Pulmonary resection for sarcoma of the lung was apparently first achieved by Lilienthal[5] in January, 1933, but the patient died on the fourth postoperative day and necropsy proved the pulmonary lesion to be a metastatic mass. In July, 1933, Archibald[1] successfully performed a pneumonectomy on a patient with a sarcoma of the upper lobe and, in October, 1933, Rienhoff[8] removed the left lung of a 3-year-old child who had a fibrosarcoma. So far as we are aware, our patient represents the first case of primary leiomyosarcoma of the lung successfully treated by pneumonectomy. In a recent review of primary pulmonary sarcomas,[7] we discovered reports of 15 cases of leiomyosarcoma of which 7 were resectable.

This case exemplifies several cardinal points in the diagnosis and management of pulmonary tumors.

1. Persistent hemoptysis calls for complete clinical, roentgenologic, and bronchoscopic examinations.

2. Extensive atelectasis may result from small tumors and even complete atelectasis of a lung does not mean that resection may not be successful.

3. A neoplasm may involve the bronchus quite close to the trachea. Successful excision may require complete removal of the bronchus and, at times, even a portion of the trachea.

4. Surgical removal of pulmonary sarcomas is desirable and, as in our case, may result in a long and useful life.

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SINGLE DOSAGE X-RAY RADIATION TO THE LUNG AND POSTERIOR MEDIASTINUM THROUGH THE OPEN CHEST

I. Results in Dogs Receiving Dosage Between 6,000 and 15,000 r

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IN 1912, Torek[6] performed the first successful esophagectomy for carcinoma of the esophagus. Twenty years later, Graham[3] was able to do a pneumonectomy for carcinoma of the lung with long-term survival. Even with subsequent advances in the treatment of these two diseases the prognoses are not good. The resectability rate in cancer of the lung is variously given as 29 per cent[4] and 41 per cent[2] and the 5-year survival figure as 5.3 per cent.[4] Of the patients having a resection, 31.7 per cent[2] were alive at the end of 5 years. The 5-year survival rate for carcinoma of the esophagus varies between 0 and 5 per cent.[5] Another series is more encouraging.[7] Here 6 per cent of the patients operated upon for midesophageal carcinomas and 15 per cent of those operated upon for lesions at the lower end of the esophagus were alive after 5 years. If the tumor was localized or metastatic only to regional lymph nodes, the rates, respectively, became 23 and 32 per cent. These percentages fall sharply if based on the total number of patients in the series. Both conventional x-ray therapy and chemotherapy have had trials but at the present time they offer only palliation in selected cases in most instances.

The dismal situation of low resectability and cure rate for carcinoma of the lung prompted Chesney and Daughtry[1] to radiate the lung through the open chest at the time of thoracotomy in some of the patients that they deemed nonresectable. As yet, their experience is meager and their approach has necessarily been cautious.

PURPOSE AND METHOD

Information on single dose x-ray radiation administered directly to the lung or mediastinum without the interposition of the chest wall is lacking. A long-term study was undertaken to investigate this modality of therapy because of possible direct clinical application. Patients with nonresectable tumors of the lung, esophagus, or posterior mediastinum might be candidates for this type of treatment. Patients whose tumors had been resected, but in whom there were positive regional lymph nodes, might also benefit from such therapy. The purpose of the investigation, then, was to establish the maximum safe

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Received for publication Dec. 2, 1957.

dose of x-ray radiation that may be given by this method, to estimate if such doses were cancerocidal, to determine if they produced less systemic effect than conventional forms of therapy, and to determine what complications can be expected. It was also the aim of the study to search for means of eliminating or treating complications. Finally, assuming that a suitable technique was developed and that doses were cancerocidal, information about pulmonary function before and after treatment must be gathered.

Mongrel dogs weighing between 9 and 27 Kg. were used. The state of health of the animals varied considerably but there was a coarse correlation between the hemoglobin and white cell count with the clinical condition of the dog. In south Florida, many of the mongrel dogs are underweight, many have heart worms, and most are infested with intestinal parasites. Under intravenous veterinary Nembutal anesthesia, the dogs' tracheas were intubated and connected to a respirator. A thoracotomy in the right fourth interspace was performed under sterile conditions. A circular cone made of sterile sheet lead was inserted into the posterior aspect of the wound in such a way that the tissues surrounding the portal of radiation entry were completely protected by at least 2 mm. of lead. The entry portal measured 28.26 sq. cm. In one group of dogs, the radiation was directed through the lung toward the posterior mediastinum. In this group, care was taken to see that there were no atelectatic areas during radiation. In the second group, the lung on the side that was operated upon was packed away from the entry portal, thus allowing the radiation to pass directly to the mediastinum without going through the lung. Following administration of radiation, the chest was closed in layers using continuous sutures of silk. Some of the dogs received antibiotics but in this series it neither affected the survival time nor the complication rate. Postoperatively, the dogs were fed standard kennel rations and were observed for symptoms. Hemograms and chest films of the animals were obtained at intervals. At the time of death, post-mortem examinations were done on all dogs.

The dose of radiation delivered varied but the factors of distance and quality remained constant. The radiation used was generated by a self-rectified therapy unit operated at 250 PKV, 30 Ma., at surface-target distance of 25 cm. and with an output of 1,200 r per minute in air at the surface of entry. The quality of the entry radiation was 4.0 mm. aluminum half-value layer. In vivo determinations and measurements made of the isolated respiring lungs of a dog showed a depth dose in the mediastinum at the level of the esophagus to average 65 per cent of the entrance surface dose when the radiation passed through the lung. The periphery of the opposite lung received 40 per cent. The absorbed dosages are expressed in RADs (radiation absorbed dose) and in tissue roentgens at the depth in question.

RESULTS

The dogs have been divided into 2 groups. Those receiving radiation through the lung have been designated as Group I and have been further

subdivided according to the amount of radiation they received. Group II comprised those animals that received radiation directly to the mediastinum with the lung protected. Statistically the 2 groups did not lend themselves for comparison or was such an analysis of the subgroups possible. Certain observations were, however, valid. Tables I, II, and III record the gross postmortem findings of the first group and Tables IV and V do the same in those dogs receiving radiation directly to the mediastinum. No effort was made to estimate accurately the body surface of the animals but the tables do record the body weights. Survival time, complications, and clinical course did not correlate with the body weights.

TABLE I*

DOG	WEIGHT (KG.)	HGB.	W.B.C.	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
2	11.8	--	--	10	+	++++	0	+++	++
4	14.0	--	--	42	++++	+++	++	0	0
7	13.6	10.2	11.200	103	†	+++	++	?	?
8‡	18.1	--	--	10	++	+++	+++	+	+
11§	18.1	9.2	10,000	60	0	++++	++++	+++	+
16	15.4	8.6	7.450	32	0	+++	+++	+++	+++

**Group I*—Dogs receiving a single exposure of 12,000 r in air to the exposed lung surface and a depth dose of 7,800 r (7,254 RAD) to the esophagus.

†250 c.c. fresh blood.

‡This dog received 15,000 r.

§Heart showed reaction to radiation.

TABLE II*

DOG	WEIGHT (KG.)	HGB.	W.B.C.	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
9	11.8	10.6	11,300	27	++++	+++	0	+++	+++
10	22.7	9.9	15,300	25	++	++++	+++	0	0
12	15.9	9.9	18,000	16	++++	++++	0	+++	0
13	15.9	9.0	7,300	26	++++	+++	++	++	+
14	15.9	13.9	10,750	27	+	+++	+++	+++	+++
15	15.9	11.0	13,700	23	+	++++	++	++	+

**Group I.*—Dogs receiving a single exposure of 10,000 r in air to the exposed lung surface and a depth dose of 6,500 r (6,025 RAD) to the esophagus.

TABLE III*

DOG	WEIGHT (KG.)	HGB.	W.B.C.	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
1	17.7	--	--	13	†	0	++	+	+
3‡	11.3	--	--	240+					
5	25.0	--	--	13	§	+++	+++	0	0

**Group I*—Dogs receiving a single exposure of 8,000 r in air to exposed lung surface and a depth dose of 5,200 r (4,836 RAD) to the esophagus.

†Pleural fistula, wound not well protected.

‡Received 6,000 r; dog still living.

§Empyema.

The hemoglobin in milligrams per cent, and the white blood cells per cubic millimeter were recorded. These determinations were done at the time of thoracotomy and then at weekly intervals. Those in the tables are the initial values. As has been stated, the hemoglobin and white count reflected the

TABLE IV*

DOG	DOSAGE OF RADIATION	WEIGHT (KG.)	HGB.	W.B.C.	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
18	15,000 r	13.6	8.4	9,000	3	++++	0	+	++	+
19	15,000 r	18.6	9.6	22,700	14	+	0	++	+++	+++
20	15,000 r	18.6	8.4	16,000	12	0	0	+++	+++	+++
21	15,000 r	11.3	6.6	7,900	4	+++	0	+++	0	0
22	12,000 r	9.5	7.9	6,600	12	++	0	0	+++	0
23	12,000 r	10.4	9.7	22,800	20	0	0	+++	+++	+++

**Group II*—Dogs receiving a single exposure of 12,000 to 15,000 r (11,160 to 13,950 RAD) to the depth of the esophagus with the lung shielded.

TABLE V*

DOG	DOSAGE OF RADIATION	WEIGHT (KG.)	HGB.	W.B.C.	SURVIVAL IN DAYS	PLEURAL FLUID	REACTION TO LUNG	PNEUMONITIS	MEDIASTINAL REACTION	ESOPHAGITIS
24	8,000 r	--	9.8	14,500	6	+	0	++	+	0
25	8,000 r	9.0	10.2	6,200	64	+	0	++++	?	?
26†	8,000 r	27.2	19.1	11,600	100+‡					
38	6,000 r	15.0	--	--	2	0	0	++	0	0

**Group II*—Dogs receiving a single exposure of 6,000 to 8,000 r (5,680 to 7,440 RAD) to the depth of the esophagus with the lung shielded.

†Greyhound.

‡Still living.

general health of the animal with some accuracy. During the postradiation (and postoperative) course there was no recognizable change in the hemoglobin for all the dogs or for any group or subgroup. Generally speaking, if the animal was malnourished and had a low hemoglobin it rose as a result of the routine kennel ration that he received. If the animal developed esophagitis and stopped eating, the hemoglobin fell. The radiation had no effect on the hemoglobin in the animals recorded in the tables. Similarly the white blood cell counts that were listed were the initial values. Here too there was no discernible pattern. If the dog developed pneumonitis from the radiation with secondary infection, the white count rose; otherwise, it remained unchanged. In no subject did a leukopenia develop.

The x-ray radiation dose within the range administered did not affect the survival time. Thus, the dogs receiving 6,000 r did not routinely live longer than those receiving 15,000 r. Because of the small size of the groups this does not have statistical significance. Similarly no difference in survival time existed if the dog received the radiation directly to the mediastinum or through the lung. Two dogs survived. One was a greyhound in excellent physical condition at the time of thoracotomy. At post-mortem examination many of the dogs had significant pleural effusions. Those that were graded four plus had between 700 and 900 ml. of fluid present in the pleural spaces. By far the greater amount of fluid was present on the right, which was the side of thoracotomy, but often fluid was also present on the left. This was understandable when it was realized that the left lung received 40 per cent of the radiation administered to the surface of the right. As a general rule those dogs that had

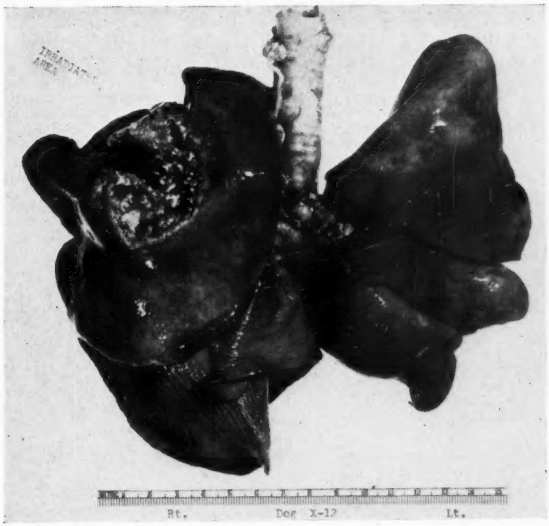

Fig. 1.—A photograph of the lungs of Dog 12 (Table II) shows necrosis of the radiated lung with excavation. The left lung and the remainder of the right lung display only minimal changes. This dog received 10,000 r directly to the lung 16 days before death.

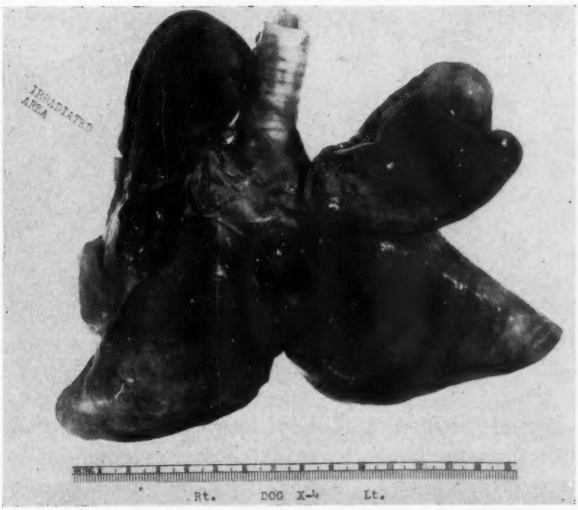

Fig. 2.—A photograph of the lungs of Dog 4 (Table I) shows radiation reaction less intense than that in Fig. 1. This animal received 12,000 r directly to the lung 42 days before death.

radiation through the lung (Group I) had more pleural effusion at autopsy than did those that received it directly to the mediastinum (Group II). Almost all of the animals developed some pleural effusion within the first week following radiation as indicated by serial x-ray studies. In addition to a massive pleural effusion, Dog 4 had a pericardial effusion large enough to cause death from tamponade. This occurred early in the series and all subsequent dogs had windows cut in the pericardium.

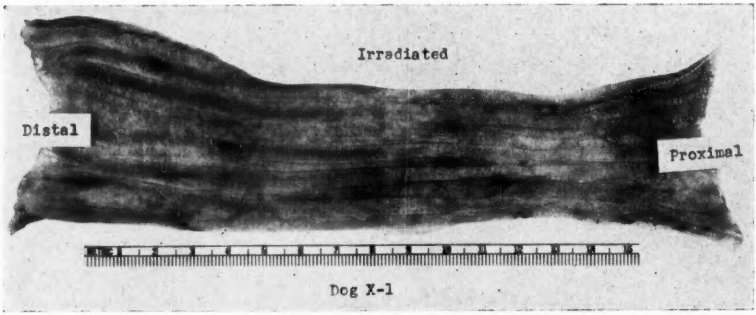

Fig. 3.—A photograph of the esophagus of Dog 1 (Table III) shows mild erythema in the radiated area. Radiation was through the lung.

Fig. 4.—A photograph of the esophagus of Dog 20 (Table IV) shows an intense reaction with sloughing of the mucosa in the radiated area.

The reaction of the directly radiated lung in Group I varied. Pulmonary reaction in the dogs receiving the higher dosage was not uniformly greater than in those receiving 6,000 r. Fig. 1 shows the typical gross appearance of the lung in Group I dogs that were graded four plus. Fig. 2 shows a less intense reaction. On serial x-ray studies the lung reaction appeared as a circumscribed density within the first week and persisted on subsequent films until the animal finally died.

"Pneumonitis" in the tables referred to changes in the left lung and in areas of the right lung that were not directly within the field of radiation. Pneumonitis was used here to mean pneumonia, atelectasis, or pulmonary edema. Usually all three were present in varying degrees. Chest films showed pneumonitis within the first 7 days but these x-ray findings did not persist even if considerable pathologic change was found at autopsy.

Mediastinal reaction consisted of edema, induration and later fibrosis of the posterior mediastinum. Changes here paralleled the esophagitis that developed but occurred earlier. The esophagitis was always more intense in the Group II dogs and in some instances the trachea and bronchi were the site of severe reactions. Fig. 3 shows only minimal changes in the esophagus of a dog receiving radiation through the lung. Fig. 4 demonstrates mucosal sloughing in a dog receiving radiation directly to the mediastinum. Esophageal perforation or fistula did not occur. Clinically the animals that developed esophagitis stopped eating about 10 days following radiation. Many of them developed a cough that was due to an associated tracheitis, bronchitis, or both. Some dogs then improved only to stop eating again a day or 2 before death.

The clinical course of the 2 groups was slightly different. All dogs recovered well from thoracotomy and radiation. On the first postoperative day they exhibited little or no discomfort, walked about and ate and drank well. The dogs in Group I for the most part did well until one or 2 days before death. Those in Group II also did well initially but about 10 days postoperatively they developed signs of esophagitis and tracheitis as manifested by cough and failure to eat. Often there would be transient improvement only to be followed by a recurrence of symptoms which shortly preceded death.

DISCUSSION

Single dosage x-ray radiation to the lung and posterior mediastinum through the open chest was a simple procedure. The radiation was immediately completed rather than prolonged. Systemic manifestations, such as radiation sickness, were absent with this method of administration. Anemia and leukopenia did not develop. With the protection used, there was no delay in wound healing and no skin damage. All of these animals received much larger than calculated cancerocidal dosages of x-ray radiation through a relatively large port. With the dosage used, it was not surprising that all animals (except 2) died. Death was due to direct destruction of tissue or a complication of tissue damage. Thus mortality was from pleural effusion, pneumonitis, or esophagitis in almost all instances. Possibly some of the dogs would have survived longer had their complications been treated. Because of the much higher than cancerocidal doses used in this series, no conclusions regarding clinical application can be made. Further laboratory work with lower but still cancerocidal dosages is indicated. A different technique of radiation administration with or without the addition of a chemotherapeutic agent bears investigation.

SUMMARY AND CONCLUSIONS

Through a thoracotomy, one group of dogs received single rather high doses of x-ray radiation directly to the lung while a second group received it to the posterior mediastinum. In the first group, death was the result of pleural effusion or pneumonitis. In the second group, esophagitis with or without tracheitis caused death. Further investigation is warranted using lower but still cancerocidal doses of radiation.

The assistance of Drs. John B. Miale and Raymond E. Parks is gratefully acknowledged.

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HYDATID CYST OF THE LUNG WITH BRONCHOGRAPHIC EVALUATION OF TREATMENT BY INTERNAL SUTURE OF THE PERICYST

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HYDATID disease (echinococcosis) is a common parasitic infection in certain sheep- and cattle-raising areas. South America, Australia, New Zealand, North and South Africa, and the Middle East are particularly affected. Cases from these endemic regions will appear elsewhere with greater frequency as world travel increases and it is also possible for other endemic foci to occur wherever conditions are favorable to the parasite. Thus, it is likely that the clinical features and treatment of various aspects of this disease will be of more general interest in the future. For this reason we are presenting our experience with cases of hydatid cyst of the lung in the American University Hospital during the past 10 years (July 1, 1947 to June 30, 1957).

PARASITOLOGY

Before dealing with clinical problems, a brief review of the life history of the responsible parasite may be helpful. Hydatid cysts are the larval stage of the minute tapeworm, *Echinococcus granulosus,* which infests the intestine of dogs and dog-like animals (the definitive hosts). The worm, which is only 2 to 8 mm. in length, consists of a scolex and neck followed by three or four successively larger segments. The terminal segment, or proglottid, contains eggs which are passed in the dog's stools to contaminate the food or water consumed by certain herbivorous mammals such as cattle, sheep, and human beings (the intermediate hosts). Human infection, especially in the case of children, may also result from such intimate association as playing or living with dogs. When the eggs are ingested by an intermediate host, they hatch in the upper gastrointestinal tract to release a larval embryo which penetrates the bowel wall. The embryo then enters the portal circulation or, less frequently, the lymphatics or systemic veins. Most of the embryos will be filtered out in the liver with the lungs being the second most commonly affected organ. Embryos may occasionally escape these two filters and lodge in other parts of the body. Multiple organ involvement is not uncommon.

After the larval embryo has come to rest, and if it is not destroyed by the humoral and tissue reactions of the host, it develops into an echinococcus cyst. This lesion is appropriately called a hydatid or a hydatid cyst since the

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term "hydatid" is derived from a Greek root meaning "watery vesicle." The growth of the cyst is slow and insidious, the rapidity depending on the environment. At the end of 6 months it will usually measure only 1 to 2 cm. in diameter but eventually it may reach a huge size and contain several liters or more of fluid. Pulmonary cysts larger than 20 cm. are not common but hydatids filling an entire hemithorax are occasionally seen. Barrett and Thomas[1] have noted cysts to grow to 6 cm. diameter in the lung within a year. It is also frequently observed that a cyst may remain fairly constant in size for several years at a time.

Table I gives the organ distribution in the 176 patients with hydatid cyst seen at the American University Hospital during the past 10 years. The diagnosis in all cases was proved by operation or autopsy. The relative incidence of pulmonary cysts in our experience (32 per cent) is somewhat higher than that usually reported. For example, Dévé[4] noted liver cysts in 75 per cent of cases and pulmonary cysts in 10 per cent. Chandler[2] records a 60 to 75 per cent liver involvement as compared with a 20 per cent lung involvement. On the other hand, Craig, Faust, and Miller,[3] state that in Chile pulmonary hydatid is as common as hepatic hydatid. It is most probable that, instead of a varying predilection of the parasite, the composition of statistics from different institutions is influenced by extraneous factors.

TABLE I. ORGANS INVOLVED IN 176 CASES OF HYDATID CYST

CASE NO.	LIVER	LUNG	SPLEEN	KIDNEY	HEART	PERITONIUM	MISCELLANEOUS
A. Distribution of 164 Cases of Hydatid Cyst With Involvement of a Single Organ							
	84	59	3	3	1	3	11*
B. Distribution of 12 Cases of Hydatid Cyst With Multiple Organ Involvement							
1	x	x					
2	x	x					
3	x		x				
4	x		x				
5	x			x			
6	x			x		x†	
7	x					x†	
8	x					x†	
9	x						
10		x	x				
11		x†			x		
12			x	x			

*Soft tissues 4, orbit 2, spine 2, brain 2, thyroid 1.

†These were unquestionably secondary cysts due to rupture of a primary cyst in the other organ listed.

An understanding of the anatomy of the hydatid as shown in Fig. 1 is important. The parasite itself is a fluid-filled cyst, the wall of which is composed of an opaque, white, friable membrane that is paper-thin during early development and eventually reaches only a few millimeters in thickness. The cyst wall is actually composed of two layers—an inner germinal and an outer acellular layer which is laid down in concentric laminae by the internal germinating surface as a protective covering around itself. The germinal membrane secretes a water-clear hydatid fluid into the cyst cavity which is thereby tensely distended to a globular shape. The germinal surface, if it is fertile, produces

small, thin-walled blisters or pedunculated cysts on its inner surface. These are known as *brood capsules* and in them are formed tiny scolices which may number several million in a large cyst. Detached and free-floating brood capsules and scolices make up a fine sediment called *hydatid sand* in the cyst fluid.

If the germinal membrane itself either invaginates or evaginates, a portion of it may be pinched off to form a *daughter cyst* which has the characteristic laminated membrane, hydatid fluid, brood capsules, and scolices. These daughter cysts may also develop from brood capsules or scolices. The daughters are usually inside but may be outside the mother cyst membrane the continuity of which is broken by the irritant or aging process which has stimulated the daughter cyst formation.[5] Daughter cysts are fairly common in hydatids of the liver but are infrequent in the lung. In our 77 pulmonary hydatid cysts, only 3 had daughter cysts.

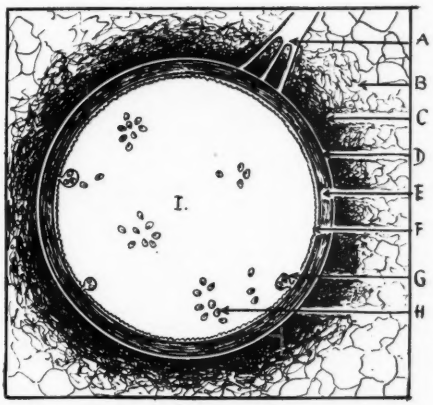


Fig. 1.—Schematic representation of a simple primary hydatid cyst of the lung. *A*, Bronchi. *B*, Lung parenchyma. *C*, Pericyst (or adventitia). *D*, Potential space between pericyst and parasite. *E*, Laminated layer of parasite membrane (ectocyst). *F*, Germinal layer of parasite membrane (endocyst). *G*, Brood capsule containing scolices. *H*, Free-floating scolices. *I*. Hydatid fluid.

Both cyst fluid and scolices are a particular menace. Due to the fact that the host develops more or less antibodies in response to the parasite, the leakage of fluid into the tissues after spontaneous or surgical rupture of the cyst wall may produce any of the whole gamut of immunologic reactions from urticaria to fatal anaphylactic shock. The scolices, which readily escape as the fluid leaks, will implant themselves and produce *secondary hydatid cysts* which are so called to distinguish them from the *primary hydatid cysts* that arise directly from ingested ova. Because of the risks of anaphylaxis and implantation, spillage of hydatid fluid must be carefully avoided during surgical treatment.

The hydatid is not attached in any way to the host but receives all its nourishment by osmosis through the cyst membrane. As the cyst grows, the host tissues are compressed to form an encircling, fibrotic sheath or adventitia

which in the larger pulmonary cysts may be a half centimeter or more in thickness. This zone is known as the *pericyst* to distinguish it from the parasite itself. The expansion of the hydatid almost invariably erodes one or more small bronchi which open into the potential space between pericyst and the parasite. Fluid leaking from the hydatid may therefore enter the bronchial tree, and bacteria from the respiratory passages have access to the pericyst cavity. When pulmonary cysts rupture spontaneously, as they frequently do, both pericyst and hydatid promptly become infected. This results in death of the parasite and its scolices. An infected hydatid will no longer implant or cause anaphylactic reactions. Viable intact hydatids (whether primary or secondary) are termed *simple* cysts. Ruptured or infected hydatids are considered *complicated* cysts.

The life cycle of this sinister parasite is completed when the cyst-containing viscera of intermediate hosts, such as sheep and cattle, are eaten by dogs. The ingested scolices develop into adult tapeworms in the dog's intestines (Fig. 2).

Fig. 2.—Life cycle of the tapeworm, *Echinococcus granulosus*.

Definitive Host : *Usually the dog*

The adult tapeworm is harbored in the small bowel and ova are passed in the stools.

The adult tapeworm develops from ingested scolices when organs infected with hydatid cyst are eaten.

Ova ↓ ↑ Scolices

Intermediate Host : *Usually cattle, sheep, and man*

Ingested ova develop into primary hydatid cysts, the larval stage of the parasite; these cysts contain scolices.

↓

Secondary Hydatid Cysts

These develop in the intermediate host from implanted scolices when a primary cyst ruptures.

Echinococcus granulosus is the only hydatid-forming parasite which we have encountered in the Middle East. In Europe, Siberia, and Alaska, there is seen an alveolar form of echinococcosis characterized by the formation of multiple thin-walled vesicles which infiltrate the surrounding tissues. It has been suggested by Rausch and Schiller[7] that this alveolar hydatid disease is caused by the larval stage of *Echinococcus sibiricensis* which is a tapeworm morphologically almost identical with *E. granulosus*.

PULMONARY CYSTS AT THE AMERICAN UNIVERSITY HOSPITAL

General Data.—During the past 10 years, 62 patients with proved primary hydatid cyst of the lung have been treated. A few of the characteristics of this series follow.

Total patients: 62 of which 4 were in the age range of 0-10 yr.; 13, 11-20 yr.; 16, 21-30 yr.; 11, 31-40 yr.; 12, 41-50 yr.; 4, 51-60 yr.; 2, 61-70 yr. The youngest patient was 5 and the oldest 65 years old.

Sex distribution: There were 44 males and 18 females.

Incidence of complicated cyst: 33 of the 62 patients had complicated cysts.

Multiple cysts: 11 patients had multiple cysts. Bilateral cysts occurred in 5 patients. The total cysts in 62 patients was 77.

Daughter cysts: Three cysts (all complicated) contained daughter cysts.

Location of 77 cysts: There were 45 (58 per cent) on the right side: upper lobe, 14; middle lobe, 11; lower lobe, 20. On the left side, there were 32 (42 per cent): upper lobe, 13; lower lobe, 19.

Size of 73 cysts recorded: 1-5 cm., 17; 6-10 cm., 48; 11-15 cm., 7; 16-20 cm., 1.

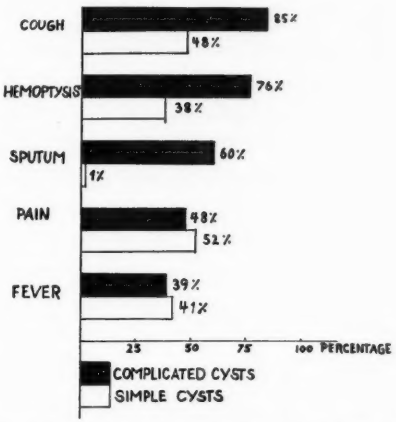


Fig. 3.—The incidence of various symptoms in 62 patients with pulmonary hydatid cyst.

Symptoms.—Simple cysts cause no complaints until pressure symptoms or pericystic pneumonitis in compressed or obstructed lung tissue occur. Seven of our patients had the diagnosis made on chest radiography or fluoroscopy done for another purpose. The incidence of asymptomatic cysts discovered in this manner will doubtless increase in the future.

Complicated cysts are the result of rupture and infection. Their symptoms vary over quite a wide range. The patient may cough up clear fluid or fragments of cyst membrane. A large membrane in the tracheobronchial tree may cause localized obstruction or even death from asphyxia. In rare instances, the cyst may rupture into the pleural space to produce pyopneumothorax, and pleural implantation of secondary cysts may follow. Widespread pneumonitis occasionally gives the picture of acute pneumonia or pulmonary abscess. More or less purulent sputum is common. On the whole, however, the

patient with a complicated cyst is not as sick as one would expect from the appearance disclosed by x-ray studies and he may even have minimal or no complaints for long periods.

The commonest symptoms of both simple and complicated cysts are cough, purulent sputum, hemoptysis, pain, and fever. The incidence of these complaints in our patients is shown in Fig. 3. It is important to note the high incidence of hemoptysis. Streaking of sputum with bright blood intermittently is most frequent but hemoptysis up to 100 ml. of blood has occurred rarely in this series. Variations between these two extremes are not unusual. The chest pain is characteristically mild or moderate and localized to the region of the chest wall near the cyst. It ranges from pleuritic to an ill-defined dull ache. The fever occurs either in acute exacerbations with symptoms suggestive of respiratory infection or as a low-grade chronic pyrexia. It is common for the symptoms of hydatid cyst to begin with an episode suggestive of pneumonia. It is surprising to find that fever has been so frequent in simple cysts;

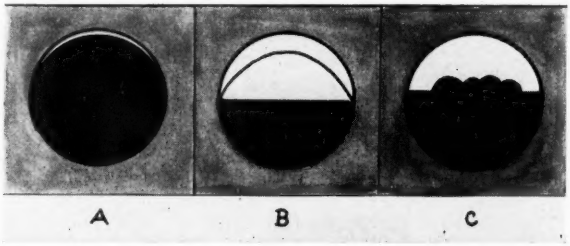


Fig. 4.—Diagrammatic representation of some typical x-ray findings (after Dévé). *A*, A tiny crescent of air may sometimes be seen between parasite and pericyst. When present it is practically pathognomonic but many cysts do not show this sign. *B*, A double space may result when air enters a ruptured hydatid. *C*, A "water-lily" sign is frequently seen in ruptured hydatid cyst. The collapsed hydatid membrane scallops the air-fluid level. Taking films with the patient in lateral decubitus or other positions may aid in visualizing the membrane.

it should be added that the duration and intensity of fever is characteristically greater in the complicated cases. The duration of symptoms prior to admission in 62 patients follows.

	Simple Cysts	*Complicated Cysts*
< 6 months	16	13
7-12 months	3	9
> 1 year	4	10
No symptoms	6	1

Radiology.—Chest radiography is essential to diagnosis unless the patient actually coughs up a membrane. Dévé[4] has summarized the roentgenographic signs in a series of diagrams of which Fig. 4 is a modification. Where echinococcosis is endemic, clinicians are generally quite familiar with the radiologic appearance of the lesion and most cases are correctly diagnosed preoperatively. The simple hydatid appears as a sharply defined oval or spherical shadow of homogeneous density in the lung field and is easily confused with tuberculoma or neoplasm. The rounded shadow of a cyst may become oval on deep inspiration (Escudero-Nemerow sign). An even more suggestive finding, when

present, is a narrow crescent of decreased density around the margin of the mass. This is due to air between the hydatid and pericyst (Fig. 4, *A*). Tomography is useful in demonstrating the crescent more clearly. When part of the fluid has escaped from the parasite, the crescent may become quite prominent. If air also enters the hydatid cyst there may be two cavities, one above the other (Fig. 4, *B*). After complete rupture of the parasite with collapse of the membrane, an abscess cavity results. An air-fluid level is frequently present, the surface of which may be wavy or irregularly scalloped due to the crumpled, retained membrane (Fig. 4, *C*). This "water-lily" sign may also be produced by necrotic debris in an abscess cavity of other origin.

Bronchography is not particularly helpful.

Bronchoscopy.—In complicated cysts, the bronchoscopist may establish the diagnosis by removing a portion of membrane which has prolapsed into an accessible bronchus. In rare instances, the entire membrane can be teased out and healing of the pericyst, without surgical intervention, becomes a possibility. Bronchial washings should be searched for membrane shreds if no gross fragments are obtainable.

Blood Tests.—The immediate Casoni skin sensitivity test and the Weinberg complement fixation test are based on immunologic reactions. If positive, then hydatid infection has probably occurred; if negative, the disease is not ruled out. False positives occur occasionally, especially with the Weinberg test. We use these tests regularly to obtain supporting evidence but the diagnosis cannot be based upon them in endemic areas. In parts of the world where echinococcosis is rare, a positive Casoni or Weinberg would be more useful.

Susman,[8] quoting the experience of Dew and others, reports the immediate Casoni reaction to be positive in 75 per cent of simple and in 92 per cent of complicated cysts. The Weinberg test is less sensitive and is said by the same author to be positive in 50 to 60 per cent of cases. Waddle[9] has reviewed 478 cases of pulmonary hydatid which occurred in Australia and New Zealand over a 30-year period. In proved cases of hydatid disease, the Casoni was positive in 57 per cent, the Weinberg was positive in 55 per cent, and one or both were positive in about 75 per cent. Our limited experience with these two tests in pulmonary echinococcosis in 49 patients is summarized as follows.

Casoni Neg., Weinberg Neg.	11 (22%)
Casoni Pos., Weinberg Neg.	13 (27%)
Casoni Neg., Weinberg Pos.	11 (22%)
Casoni Pos., Weinberg Pos.	14 (29%)

One or both of the tests were positive in 78 per cent. There was no difference in the percentage of positive tests in simple and complicated cysts in our cases.

Eosinophil count was done in 61 cases with the following result.

Normal	0- 4%	44 cases		(72%)
Abnormal	5- 9%	10	17 Cases	(28%)
	10-15%	6		
	>16%	1		

Since intestinal parasites were very common among the patients of this series it is difficult to know the relation of eosinophilia to echinococcosis in these cases. The eosinophil count is not very helpful in differential diagnosis.

TREATMENT

Hydatid cysts of the lung should be removed when they are discovered if the patient's condition warrants. Simple, asymptomatic cysts are no exception since they will grow to troublesome proportions and there is the ever-present danger of spontaneous rupture. Furthermore, surgical treatment is safe and prospects for cure are excellent.

The various types of treatment carried out on our 62 cases are indicated in Table II. We have abandoned the method of marsupialization of the cyst to the chest wall followed by open drainage of the pericyst cavity since bronchocutaneous fistula and residual abscess are troublesome sequelae. We would now advise one of the following 3 techniques of treatment depending upon the circumstances of the case: (1) pulmonary resection, (2) dissection of the pericyst, or (3) removal of the hydatid membrane and internal suture of the pericyst.

TABLE II. TYPES OF TREATMENT IN 77 PULMONARY HYDATID CYSTS WHICH OCCURRED IN 62 PATIENTS

OPERATION	SIMPLE CYSTS	COMPLICATED CYSTS	TOTAL
Marsupialization and drainage	5	4	9
Lobectomy	1	10	11*
Dissection of pericyst	1		1
Evacuation of parasite and internal suture of pericyst	33	20	53
Nondescript	2	1	3
Total	42	35	77

*Actually only 10 lobectomies have been done since, on one occasion, 2 cysts were removed in the same lobe.

Pulmonary Resection.—It is rarely necessary to do a segmental, lobar, or more extensive resection for hydatid cysts because simpler measures suffice. We reserve resection for those cases in which sepsis in a complicated cyst has destroyed a lobe, produced neighboring bronchiectasis, or otherwise made it unwise to leave behind the pericyst and surrounding tissues. This, fortunately, is not common.

Dissection of the Pericyst.—In this technique, a dissection plane is developed just outside the pericyst which is completely enucleated with the intact parasite. The bronchi opening into the pericyst and the defect in the lung are then closed with absorbable sutures. We have had little experience with this procedure but consider it most suitable for peripherally located, simple cysts of 6 cm. or less in diameter.

Removal of the Hydatid Membrane and Internal Suture of the Pericyst.—We have found this to be the easiest, safest, and most satisfactory method of

treatment in most cases. Our results, to be discussed below, confirm this opinion. There are 3 phases of this operation deserving comment: (1) the exposure, (2) removal of the parasite, and (3) closure of the pericyst.

1. *Exposure:* This is obtained by a wide thoracotomy using an anterior, lateral, or posterior (face-down) approach depending on the location of the cyst. All adhesions are divided so that the lung is completely mobilized. This permits positioning of the cyst in the middle of the operative field with careful walling-off of pleural space and wound by moist gauze. Also important, the lung surrounding the cyst relaxes easily to permit secure closure of the pericyst and then expands freely to obliterate the space previously occupied in the thorax by the mass of the cyst.

2. *Removal of the parasite:*

a. *Complicated cysts:* If the membrane is collapsed and infected, then the scolices will not be viable and no special precautions to prevent their implantation are required. The parasite is easily managed by opening the avascular peripheral dome of the pericyst, extracting the macerated cyst membrane with sponge forceps and swabbing out the accumulated pus and debris with dry gauze. The interior of the pericyst is only mechanically cleaned and may then be closed as described below.

b. *Simple cysts:* These must be handled with care in order to avoid spillage of hydatid fluid and scolices with the danger of immediate anaphylactic shock and of delayed secondary cyst formation. At this stage of the operation, a quiet operative field is most desirable and the anesthetist is requested to minimize the respiratory motions, using muscle relaxants if necessary. The pericyst is carefully incised down to the glistening white membrane of the hydatid which tends to bulge into the pericyst incision, especially if the anesthetist applies too much positive pressure. The opening in the pericyst is cautiously lengthened for several centimeters and the margins are held up and apart by Allis forceps. Protective gauze packing is moved up to the edge of the pericyst incision. A 22-gauge needle connected to a 3-way stopcock and syringe is inserted into the cyst while a fenestrated suction tip is held near the puncture site to catch any leakage of fluid. Usually from 10 to 30 ml. of hydatid fluid are aspirated so that the cyst membrane relaxes slightly without collapsing. This fluid is evacuated into a basin through a length of rubber tubing attached to the side-arm of the 3-way stopcock. Another syringe containing formaldehyde, U.S.P. (approx. 35 per cent formaldehyde) is attached to the 3-way stopcock. The formaldehyde is slowly injected through the needle which has remained in place held steadily by an assistant. The volume of the cyst is estimated and the amount of formaldehyde is used which will result in a 1 per cent solution within the cyst. This concentration should kill the scolices in 5 minutes.[4] With the fine needle still in place to avoid leakage, 5 minutes are allowed to pass at the end of which time a second and larger needle (e.g., 15-gauge, 12 cm. length) is introduced carefully into the cyst the contents of which are aspirated using a 50 ml. syringe and 3-way stopcock with rubber tubing attached to the side-arm. Again the fenestrated suction tip is held near the site of puncture in the

cyst to pick up leakage, should it occur. As the hydatid cyst is decompressed it is possible with ease to open the pericyst widely without fear of rupturing the membrane. The pericyst margins are held up and apart by Allis forceps and the collapsing membrane is gently grasped with ring-type sponge forceps and lifted into an awaiting basin. Should leakage of fluid occur, the scolices will have been killed by the 1 per cent formaldehyde which is itself of insufficient strength to be seriously toxic to the tough pericyst lining. "Formolage," as this procedure is called, is not effective when daughter cysts are present. In these cases one must rely on aspiration alone. Daughter cysts are lifted out along with other contents of the pericyst. A large kitchen spoon or ladle may be needed in these circumstances. If there is spillage of hydatid fluid in the pericyst, it may be mopped out with 1 per cent formaldehyde solution followed by saline sponging. If this is done carefully no formaldehyde will get into the bronchial tree.

It is fortunately unlikely that scolices spilled in the pericyst cavity at operation will easily take on this leathery, avascular surface which inevitably becomes secondarily infected after internal suture. It is doubtless for this reason that Susman[8] and other Australian surgeons do not take the precaution of formolage when dealing with primary cysts of the lung but merely aspirate them and lift out the collapsed endocyst with surrounding tissues well protected. We have favored formolage as an additional precaution which is simple to carry out.

Barrett and Thomas[1] are even more assiduous in avoiding hydatid fluid spillage. In primary simple cysts of intermediate size, they advise the removal of the intact parasite. This is accomplished by a wide incision in the pericyst from which the hydatid is gently expressed by the expanding lung and the air in the pericyst when the anesthetist increases the positive pressure. These authors used in the method successfully in 17 cysts. In 4 others the cysts ruptured during the process of removal. We tried this procedure in 5 patients with cysts 10 cm. in diameter or smaller. In 2 cases the hydatid suddenly and unexpectedly ruptured while being eased out. In one of these patients, the spillage of hydatid fluid onto surrounding tissues coincided with an immediate drop of blood pressure which responded to vasopressors and was considered to be an evidence of anaphylactic shock. Although it is very satisfying to get out the parasite intact, it is our impression that the danger of uncontrolled spillage of hydatid fluid is great with this technique. For those not thoroughly experienced in judging the tensile strength of the cyst membrane, it is wiser to use the less difficult method of aspiration.

3. *Closure of the pericyst:* The objective is to obliterate the pericyst cavity as completely as possible with absorbable sutures. Chromic catgut (2-0) on an atraumatic needle is very satisfactory. The bronchial openings in the base of the cavity are individually closed if possible. In a large cyst it is rarely convenient to close them all and it is unnecessary as long as the walls of the cyst are well approximated. Working inside the pericyst cavity, deep bites in the pericyst wall are taken, purse-string and interrupted sutures being used, as

seems appropriate, until the pericyst is securely obliterated. The original incision into the pericyst is finally and tightly closed with running or Connell sutures so that there is no air leak. The chest is drained with a tube brought out through an interspace and placed on water-seal. Postoperative care includes antibiotics and the usual methods to maintain complete expansion of the lungs.

RESULTS

Treatment in our cases was carried out by a number of different surgeons. In the past 5 years most operations have been performed by members of the Resident Staff. There have been no operative deaths in the 62 patients who make up our total 10-year series.

The operation of hydatid evacuation and internal suture of the pericyst has been done all over the world for a number of years. Results appear to be satisfactory but detailed follow-up studies are still needed to complete the evaluation of this procedure. Since we have used internal suture more frequently than any other technique, we have concentrated on obtaining follow-up information on its efficacy and will now report our findings.

Forty-two patients with 53 cysts requiring a total of 46 thoracotomies have been treated by internal suture of the pericyst. There were 4 postoperative complications as follows: minor wound infection, 2; bronchocutaneous fistula, 1; and cerebral air embolism, 1.

The bronchocutaneous fistula consisted of a transient slight air leak through the site of a catheter which was sewn into the center of the obliterated pericyst cavity as a temporary drain. This was done in a few cases early in this series since it was feared that an undrained pericyst cavity might flare-up as an abscess in the postoperative period. This fear has proved to be unfounded. The patient with the fistula left the hospital on the fourteenth postoperative day and the fistula closed spontaneously shortly thereafter without treatment. The cerebral air embolism was the only significant complication encountered and a permanent left hemiparesis has resulted. This complication is likely unrelated to the operation of internal suture since air may have entered pulmonary veins which bled profusely when an unsuccessful and quickly abandoned attempt was made to dissect out the pericyst.

The duration of postoperative hospitalization required in 46 thoracotomies for internal suture is given in Fig. 5. The morbidity following this procedure has been satisfactorily low. It is necessary, however, to demonstrate that functional and anatomic results are equally satisfactory before confidence in the internal suture method can be finally established. It has been stated that the pericyst cavity will not heal completely and that a residual pocket of variable size will remain to suppurate later.[6] Also, most surgeons when doing an internal suture for the first time on a complicated cyst have doubtless shuddered at the prospect of wiping the pus out of an infected pericyst cavity and then sewing it up tightly. Unquestionably, we have to thank the antibiotics for making this relatively innocuous.

It has been possible for us to follow only 26 of the 42 patients on whom internal suture operations were done. Local conditions have made long-term follow-up difficult and many of our patients who come from other countries

are inevitably lost. The functional result in 26 followed patients is summarized in Table III. No patient had significant residual symptoms requiring treatment. We know of no recurrences due to secondary cyst formation.

TABLE III. FOLLOW-UP RESULTS ON 42 PATIENTS TREATED BY INTERNAL SUTURE

DURATION OF FOLLOW-UP	SIMPLE CYST		COMPLICATED CYST	
	EXCELLENT*	GOOD*	EXCELLENT	GOOD
<6 months	4		3	1
½-1 yr.	1			2
1-2 yr.	2	2	2	2
>2 yr.	3	1	2	1
Totals	10	3	7	6
No follow-up	9		7	

*Excellent—No symptoms.
†Good—Occasional minimal cough or chest pain.

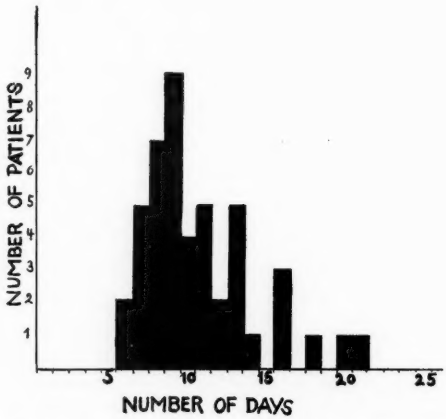


Fig. 5.—Duration of postoperative hospitalization after 46 thoracotomies for internal suture of hydatid cyst.

Anatomic results were checked in 20 of the 26 patients by bronchogram at a period varying from a few months to several years postoperatively. In 12 patients the cysts were simple and in 8 cases they were complicated. The findings follow: In a total of 20 patients there was no residual cavity or bronchiectasis in 17 patients; minimal residual localized bronchiectasis in 1 patient; and residual cavity, 1.5 cm. diameter (Figs. 6 and 7), in 2 patients.

In the 3 patients showing slight residual changes on bronchogram, the cysts were complicated and in the lower lobe. One of the patients (with residual 1.5 cm. cavity) has occasional cough and the other 2 have no symptoms.

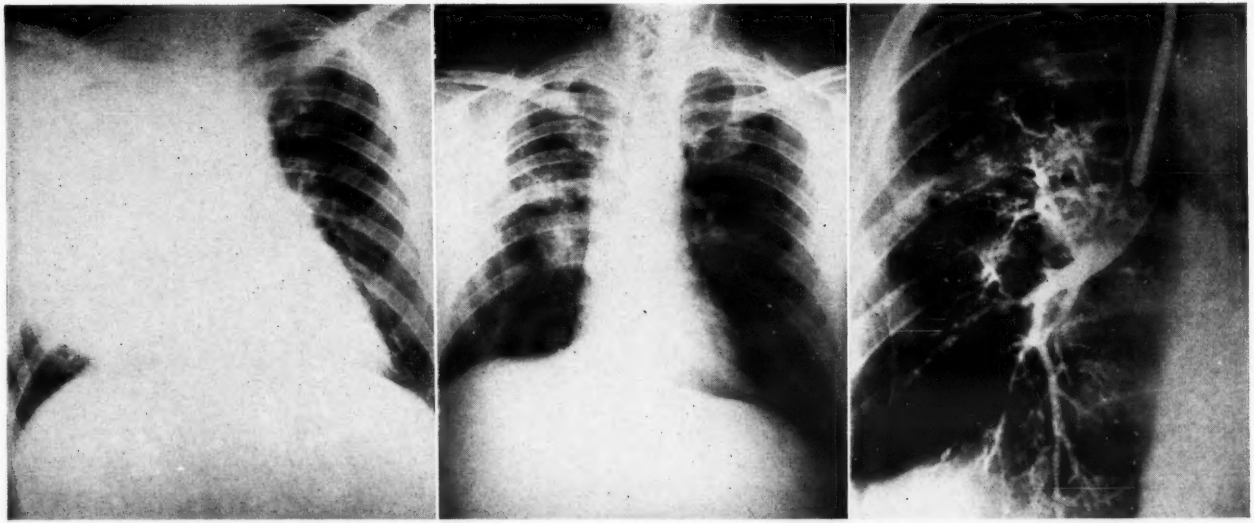
In conclusion, our studies confirm that internal suture of the pericyst is safe and effective. We could wish for longer and more complete follow-up of our patients and hope by continuing observation to arrive at a more thorough evaluation of this problem in the future. The method is applicable to both simple and complicated cysts. Even large hydatids can be well obliterated as shown by Fig. 8. Fortunately, the procedure is technically easy and allows maximum preservation of functioning lung tissue.

A. *B.* *C.*

Fig. 6.—Case No. 105960. A 52-year-old man with a 4 cm. complicated hydatid cyst in the right lower lobe. Treated by internal suture of the pericyst. Plain film and bronchogram 6 months postoperative. A 1.5 cm. residual cavity communicates with the right posterior basal bronchus. Symptoms now consist of occasional nonproductive cough. *A*, Preoperative. *B*, Postoperative. *C*, Bronchogram.

A. *B.* *C.*

Fig. 7.—Case No. 62066. A 33-year-old woman with a 6 cm. complicated hydatid cyst in the left lower lobe. Treated by internal suture of the pericyst. Plain film and bronchogram 4 months postoperative. A residual 1.5 cm. cavity communicates with the posterior basal bronchus. She now has no respiratory symptoms. *A*, Preoperative. *B*, Postoperative. *C*, Bronchogram.



A. *B.* *C.*

Fig. 8.—Case No. 116269. A 31-year-old man with an 18 cm. simple hydatid cyst of the right middle lobe. Treated by internal suture of the pericyst. Plain film and bronchogram 5 months postoperative. There is no residual cavity or bronchiectasis. He now has no respiratory symptoms. This patient has a permanent left hemiplegia presumably due to cerebral air embolism during operation (see text). *A*, Preoperative. *B*, Postoperative. *C*, Bronchogram.

SUMMARY

During the past 10 years, 62 patients with primary hydatid cyst of the lung have been treated surgically at the American University Hospital in Beirut without mortality.

Although a variety of procedures have been carried out, interest in recent years has been on treatment by internal suture of the pericyst after careful removal of the parasite.

Forty-two patients have had cysts treated in this manner with low morbidity and minimal complications. Follow-up examination of 26 of these patients disclosed that none had significant residual symptoms. Bronchograms on 20 of them showed that only 3 had either slight localized bronchiectasis or minimal residual cavitation.

Long-term follow-up of greater numbers of patients treated by internal suture is necessary before a thoroughly objective study of this method of treatment will be completed. However, on the basis of present evidence, it is justifiable to recommend internal suture as suitable for most hydatid cysts of the lung.

The assistance of Miss Batishwa Badawi, School of Public Health, American University of Beirut, is gratefully acknowledged.

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PANCREATIC PSEUDOCYST OF THE MEDIASTINUM

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INTRODUCTION

IT is the purpose of this presentation to record the rare instance of pseudocyst of the pancreas presenting as a tumor of the mediastinum, and to discuss its diagnostic and therapeutic considerations.

CASE REPORT

History.—A 41-year-old white, married, used-tire dealer entered the hospital complaining of mid-back pain and easy fatigability of 6 weeks' duration. The pain was mild, recurred almost daily, lasted several hours at a time and was unrelated to meals or type of food ingested. On some occasions exercise relieved the pain.

At the onset of his illness 3 months earlier the discomfort was different. He experienced sharp, anterior left chest pain radiating through to his mid-back and to his shoulders. It was more intense with deep breathing. This pain disappeared in 24 hours. An electrocardiogram taken at the time was normal. There was no recurrence of this type of chest pain. Later that month an attack of left flank pain was believed to have been the result of a renal calculus. A stone was not found at cystoscopy.

There were no complaints of cough, night sweats, fever, sputum, or hemoptysis. There was no precordial pain with exertion and no symptoms involving the upper extremities. He reported that he had lost 35 pounds over a period of 7 months.

In reviewing the past history, the patient reported that 3 years earlier, after a night of heavy drinking, he had been admitted to another hospital with severe epigastric pain, nausea, and vomiting. After x-ray studies, emergency surgery was performed. The discharge diagnosis was perforated peptic ulcer. Following this procedure, he had had no gastrointestinal symptoms or recurrence of epigastric pain.

His habits included moderate daily alcohol intake, primarily whiskey, 1½ packs of cigarettes daily, and no medications. He reported no food intolerance or allergies. The family history was noncontributory.

Physical Examination.—The patient was a well-developed, well-nourished, ambulatory, white male in no acute distress. Blood pressure was 160/100 mm. Hg; temperature, 99° F.; pulse, 88; respirations, 20. Examination of the chest revealed a palpable friction rub over the left lower chest, anteriorly, along with tenderness to percussion over the tenth to twelfth ribs in the left midaxillary line. This rub was not elicited after admission. There were no other adventitious sounds. The heart was normal. The liver was not palpable. There was a right upper quadrant scar. The remainder of the physical examination was within normal limits.

Laboratory and X-ray Data.—Detailed hematology, blood chemistry, urine and stool studies were normal with the exception of the sedimentation rate of 34 mm. per hour.

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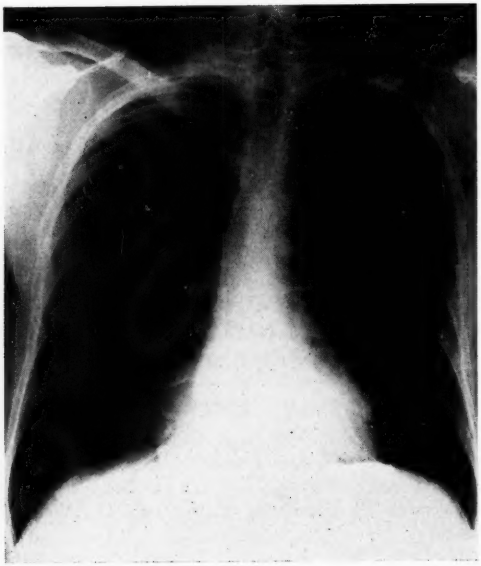
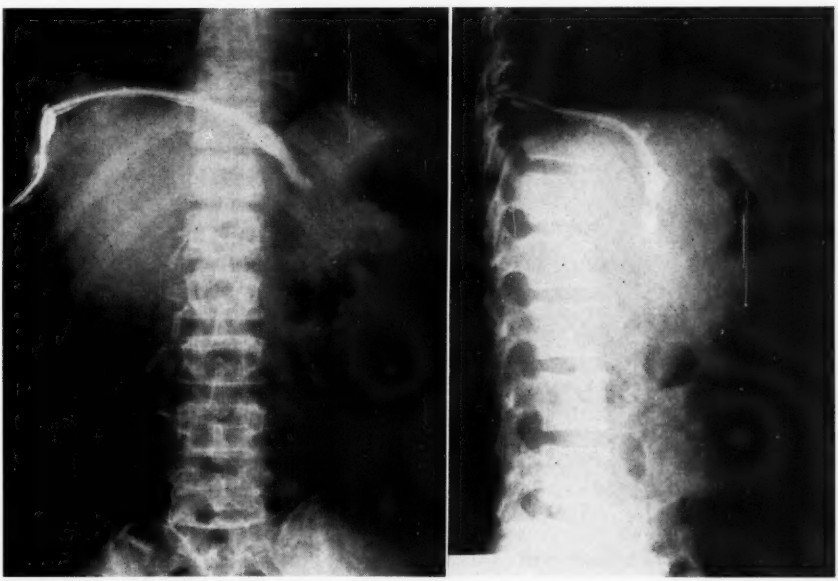


Fig. 1.—A preoperative chest x-ray demonstrating a mass in the posterior, inferior mediastinum.

Fig. 2.—Retrograde pancreatogram, taken during right thoracotomy, illustrating the subdiaphragmatic portion of the pancreatic pseudocyst and the dilated duct of the tail and the body of the pancreas.

X-rays of the chest revealed a large, globular soft tissue mass in the lower mediastinum posteriorly, located above the diaphragm on both sides of the spine. Repeated chest x-rays proved that the mass was increasing in size (Fig. 1). Esophagrams were negative for intrinsic lesions and diaphragmatic herniae. There was deviation of the esophagus to the left without impingement upon its lumen. The gastrointestinal series showed the duodenum to be irritable with a pressure defect noted within the duodenal sweep which was in itself not enlarged. Angiography did not opacify the aorta sufficiently to delineate the vascular structures from the mass. However, thoracic aortography clearly showed the aorta to be deviated by the lesion. A bone survey was negative.

Operations.—The preoperative impression was cystic tumor of the posterior mediastinum. Exploratory thoracotomy was carried out through the bed of the right eighth rib. A supradiaphragmatic cystic lesion, 8 by 5 by 4 cm. was found to the right of the lower third of the esophagus. It was densely adherent to the posterior aspect of the pericardium, and a portion of the cyst extended below the diaphragm, through the aortic hiatus. The cyst contained thin, muddy-brown fluid, small gray amorphous particles, and an amylase content of 3,050 units. The supradiaphragmatic component of the pseudocyst



A. *B.*

Fig. 3.—Retrograde pancreatogram taken on the fourteenth postoperative day ([*A*] anteroposterior and [*B*] lateral views).

was excised. The subdiaphragmatic extension was closed around a No. 18 Foley catheter by means of a purse-string suture. Urokon (30 per cent) was instilled through the catheter and was visualized in the region of the tail and body of the pancreas (Fig. 2). A diagnosis of pancreatic pseudocyst with mediastinal extension was made. Transduodenal sphincterotomy was decided upon. The thorax was closed in layers. The Foley catheter and intercostal tube were brought out in the midaxillary line.

The condition of the patient after turning him onto his back permitted abdominal exploration and definitive therapy. An ample upper abdominal right rectus incision was made. There was no free fluid in the peritoneal cavity. The gall bladder, stomach, and

duodenum were normal to palpation. The pancreas was harder and more nodular than normal. Exploration of the lesser sac revealed no evidence of a cyst. The Foley catheter could be palpated retroperitoneally. A transduodenal sphincterotomy was performed, followed by cholecystectomy. A polyethylene tube was threaded up the pancreatic duct and dye was injected for a pancreatogram. The pancreatogram was imperfectly performed. It was not possible to delineate communication between the cyst and the pancreatic duct system. The distal end of the polyethylene tube was then passed up the common duct and brought out through the cystic duct stump. A short limb T-tube was inserted into the common duct and brought out through a stab wound with the pancreatic duct catheter and with the Penrose drain placed at the foramen of Winslow before closing the abdomen in layers. During the 5-hour procedures, 1,200 c.c. of blood and 1,000 c.c. of 5 per cent glucose in water were given.

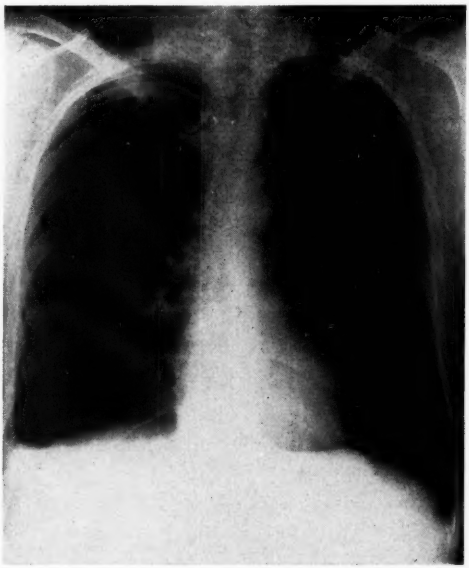


Fig. 4.—Chest x-ray one year after removal of mediastinal mass.

Postoperative and Follow-Up Courses.—The patient did well postoperatively. The pleural drainage tube was removed on the third postoperative day.

There was little drainage from the Foley (pseudocyst) catheter or from the pancreatic duct catheter. X-rays of simultaneous injections of contrast media through these catheters showed no residual pseudocyst, and no dilatation of the pancreatic duct system, as had been noted the day of surgery (Fig. 3). These catheters were removed. T-tube cholangiography showed no reflux into the pancreatic duct. The T-tube was removed.

The patient has been seen periodically for 14 months since surgery. His only symptom is fatigue. His appetite is normal. His diet is not restricted. His weight is steady at his normal 165 pounds. The physical examination is normal except for well-healed scars. His history, physical examination, and gastrointestinal x-rays give no indication of recurring pseudocyst (Fig. 4).

DIFFERENTIAL DIAGNOSIS

In the list of tumors of the posterior inferior mediastinum[1] are neurilemmoma and the less common ganglioneuroma, which are characterized by few

symptoms, if any, and the x-ray appearance of a dense rounded paraspinal mass with a sharp border and, rarely, bony erosion of the rib or enlargement of the intervertebral foramen. Esophageal neoplasms, epiphrenic diverticula,[2] and hiatus herniae frequently are suspected by symptoms and confirmed by barium meal contrast x-ray studies. They appear on the left side. Pneumoperitoneum may help differentiate diaphragmatic defects and herniation of abdominal viscera. Gastroenterogenous cysts,[3] duplications of the alimentary tract,[4] meningocele,[5] sequestered lung,[6] lipoma, pheochromocytoma,[7] and displaced kidney[8] are rare and tend to elude definitive diagnosis preoperatively, despite studies of known parameters.

Teratomas occur anteriorly.[1] Pericardio-coelomic cysts[9] are rare, especially posteriorly. Tuberculous adenitis is usually in the hilar region. Tumors of lymphoid tissue are likely to have coexisting enlargement in the more classical locations. The rare mesenchymal tumors (hemangioma, lymphangioma, pericytoma) defy diagnosis prior to histologic study. Aortic aneurysm usually appears on the left side and may be visualized with contrast media.[10, 11] Exotic hepatic infections[12] usually have abdominal findings. Lung tumors suggest the diagnosis by history and endoscopy.

Pseudocysts[15] of the pancreas are considered following the history of recurrent pancreatitis with epigastric and/or back symptoms, and an epigastric mass of inconstant or enlarging size. Pseudocyst of the posterior mediastinum is likely to elude diagnosis unless an abdominal component of the pseudocyst is recognized. Definitive diagnosis follows demonstration of pancreatic enzymes in the fluid and communication between pancreas and the mediastinal mass.[15] The patient reported here lacked the classical history of pancreatitis and had no abdominal mass. The succession of chest pain, flank pain, and back pain may have indicated extensions of the pseudocyst.

Needle puncture of a mediastinal mass is ill advised if the mass contains tumor or infected tissue, especially when the possibility of transgressing the pleural space exists. Injecting contrast media is not without hazard of extravasation or irritation (e.g., into the subdural space). Aspirated fluid of high amylase content is not specific for pancreatitis. It has been found in pleural effusions existing with primary carcinoma of the lung, and retroperitoneal primary and secondary tumors invading the thoracic duct.

When the nature of the mediastinal mass remains in doubt after appropriate studies, thoracotomy is indicated for diagnosis and therapy.

THERAPY

If the diagnosis of pancreatic pseudocyst is made through preliminary studies, surgical treatment appears mandatory to reverse the process of enlargement of the pseudocyst, and to relieve symptoms.[14] In the present case, abdominal surgery would have been performed.

One philosophy of therapy advocates sphincterotomy[14, 16] to eliminate the mechanical factors believed to be provocative of recurrent pancreatitis, affording natural route internal drainage of the cyst by the same maneuver. Earlier

forms of surgical therapy still enjoying popularity employ internal drainage by "cyst-enterostomy,"[13] or external drainage. A disadvantage of the latter is the electrolyte loss. The disadvantage of both may be the failure to obviate recurrent pancreatitis.

Technical considerations influence or compel certain surgical actions. Excision may or may not be possible. The retroperitoneal portion of the pseudocyst may be of insufficient size to permit enteroanastomosis. External drainage may be a mandatory safety valve.

DISCUSSION

In this reported case, excision of the bulk of the mediastinal mass was begun as definitive treatment but was abandoned when partial excision revealed the extent and identity of the pathology. External drainage was employed as a safety valve and as a pathway for postoperative dye injection studies. Sphincterotomy was elected to relieve the pancreatic duct obstruction believed responsible for recurrent pancreatitis and the pseudocyst. The absence of a palpable retroperitoneal mass precluded enteroanastomosis, had it been considered desirable. Cholecystectomy was performed prophylactically.[16]

All procedures were performed the same day, under the same anesthesia, to effect definitive treatment. The diagnosis appeared clear. It was believed that additional helpful data could not be obtained by other studies. The satisfactory condition of the patient before and during surgery, including turning the patient from the lateral to the supine positions, permitted this.

The immediate postoperative course, the 14-month sequel of well-being, and the radiographic evidence suggests that sphincterotomy was well chosen as therapy for this patient's problem.

SUMMARY

The case of a patient with a pancreatic pseudocyst of the lower posterior mediastinum is presented for consideration in differential diagnosis of symptomatic lesions of that area. The history and physical examination did not suggest the diagnosis.

Treatment consisted of transthoracic excision of the bulk of the cyst, and transduodenal sphincterotomy through a separate abdominal incision. Abdominal operation for internal drainage would have been performed if the nature of the lesion had been known preoperatively.

The differential diagnostic possibilities and methods have been discussed. The nonspecificity of fluid amylase content is emphasized.

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POSTINFLAMMATORY "TUMOR" OF THE LUNG

Report of a Case Simulating Malignant Thymoma

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ACUTE lower respiratory infections are occasionally harbingers of serious intrathoracic disease. Thus, many otherwise silent and unsuspected pulmonary lesions become available to roentgenologic scrutiny. There is, however, a rather rare group of postinflammatory lesions of the lung, aptly described by Umiker and Iverson,[1] which is easily confused both clinically and pathologically with neoplasm.

Herein is reported a case which presented as a rapidly growing neoplasm of the upper lobe of the right lung and was considered a malignant tumor on the initial pathologic examination.

CASE REPORT

A 3½-year-old boy was considered well until March 25, 1956, at which time he became acutely ill with a productive cough accompanied by a temperature elevation between 102° and 103° F. Treated initially as an outpatient, he was admitted to a Station Army Hospital on March 29, 1956, because of continued symptoms. Admission roentgenogram of the chest revealed a large dense shadow localized to the upper one-third of the right lung. He was diagnosed as having pneumonia and treated with penicillin and streptomycin.

Physical examination revealed an acutely ill child with a productive cough, fever of 102° F., and rather enlarged submaxillary and axillary lymph nodes. There were dullness and tubular breath sounds over the upper anterior and lateral portion of the right chest. Initial antibiotic therapy was considered ineffective. Bone marrow and routine laboratory studies were not helpful diagnostically. He was therefore transferred to Fitzsimons Army Hospital on April 3, 1956, for further evaluation and treatment.

The physical findings on transfer revealed a pale, acutely ill boy, coughing frequently and productively, with a temperature of 99.8° F. There were diminished breath sounds over the right anterior chest with slight but significant bulging in this region. No mediastinal shift was observed.

Chest roentgenograms taken at Fitzsimons Army Hospital and compared with those previously taken revealed an enlarging density in the anterior right chest continuous with the anterior superior mediastinum (Figs. 1 and 2). Diagnostic intravenous urogram showed no abnormalities. The admission blood count showed 6,800 white cells with 70 per cent neutrophils; hemoglobin 10 grams; hematocrit 31; and the sedimentation rate was 53.

It was the impression of the staff that this patient had a rapidly growing malignant neoplasm involving a large part of the right upper pulmonary lobe and upper anterior mediastinum. Therefore, after blood transfusion, a right exploratory thoracotomy was performed on April 7, 1956, at which time a large firm lobular tumor was found extending from the superior and anterior mediastinum into the right upper lobe which it largely replaced.

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This material has been reviewed and there is no objection to publication by the Office of the Surgeon General, Department of the Army, MEDCI, June 19, 1957.
Received for publication Dec. 2, 1957.

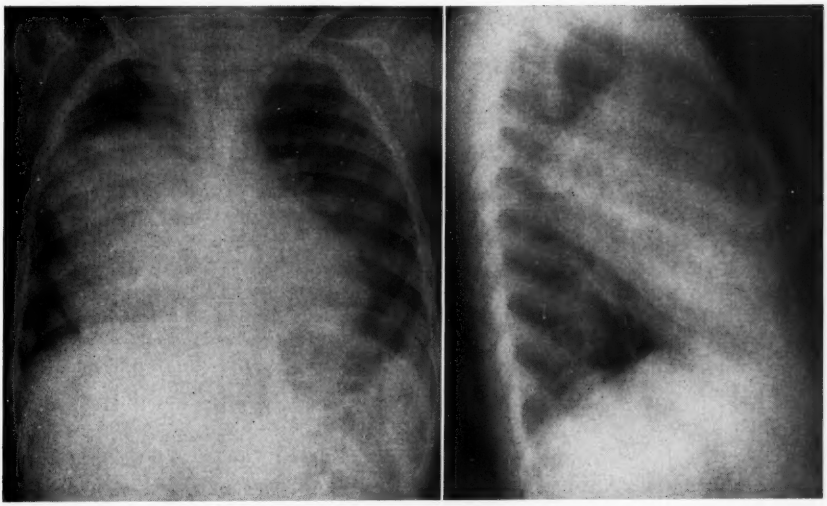

Fig. 1. Fig. 2.

Fig. 1.—Preoperative posteroanterior roentgenogram of the chest.
Fig. 2.—Preoperative lateral roentgenogram of chest.

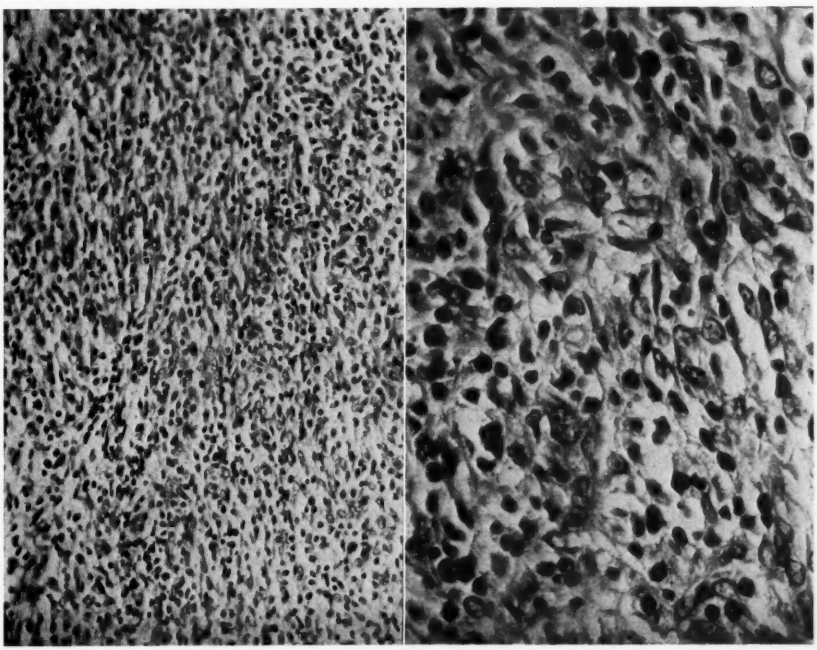

Fig 3. Fig. 4.

Fig. 3.—Low-power view showing proliferative fibroblasts with an admixture of inflammatory cells and blood vessels.
Fig. 4.—A more detailed view of the pleomorphic pattern of the lesion.

There were numerous succulent nodes about the hilus and frozen section of these revealed no tumor. Frozen section of the tumor, however, was interpreted as showing a malignant neoplasm. Grossly, it was possible to remove the entire tumor together with the right upper lobe and most of the thymus which appeared to be involved in the pathologic process.

Pathologic Report.—Grossly the tumor mass measured 10 by 7 by 5 cm. The specimen was rubbery and, upon cut section, exhibited a uniform grayish-white granular appearance with small inconspicuous areas of superimposed hemorrhage. Extension had occurred through the pleura from the parenchyma of the right upper lobe and irregularly into the mediastinal connective tissue. The thymus gland was immediately adjacent to the tumor but grossly uninvolved. Slightly enlarged regional nodes presented a normal pattern.

Microscopically, the tumor was composed predominantly of a proliferation of closely situated spindle cells with oval, vesicular nuclei arranged in irregular interlacing strands or whorls. In more cellular areas, inconspicuous fine collagen fibers and numerous but inconsistent capillaries were formed. Infrequently, production of dense collagen was seen in areas

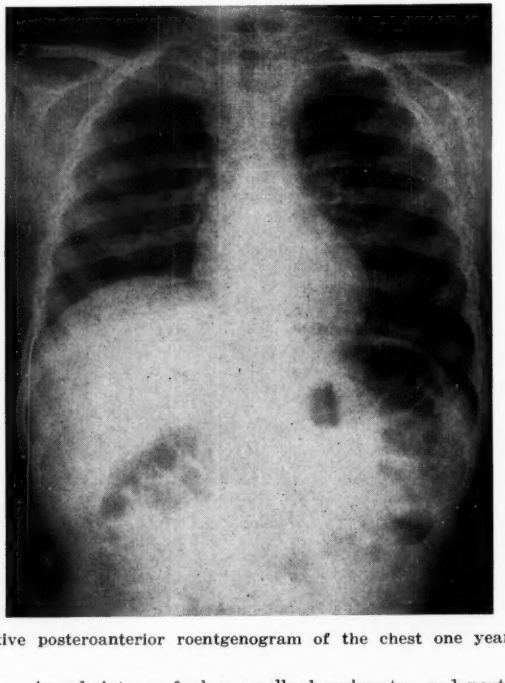

Fig. 5.—Postoperative posteroanterior roentgenogram of the chest one year after operation.

of lesser cellularity. An admixture of plasma cells, lymphocytes, and neutrophils were scattered throughout. Many of the plasma cells were immature as evidenced by enlargement of their nuclei, a homogenicity of nuclear chromatin, and loss of the eccentric position of these nuclei. A reticulum stain exhibited moderate numbers of wavy fibers which were occasionally split longitudinally, showed no lateral branching, and passed between cells. Mitotic figures were rare, occurring approximately one per high-power field. Masson's trichrome stain presented an equivocal reaction. Russell bodies were not identified, and foam cells were absent (Figs. 3 and 4). The pathologic diagnosis of a thymoma was entertained.

Postoperative Management and Course.—The patient made an uneventful recovery. Because of the initial pathologic diagnosis and upon the recommendation of the Tumor Board, a course of x-ray therapy was begun on April 16, 1956, and extended over a period of 31

days to May 17, 1956. The patient received 6 treatments a week, using 250 kv., directed through 5 by 6 cm. anterior and posterior fields covering the hilar areas and the superior mediastinum. Alternate fields received 200 r air daily for a total air dose of 2,800 r per field. The skin dose was 3,780 r, and estimated tumor dose was 2,684 r. Fourteen treatments were given to each field. The skin reaction was mild on the completion of therapy.

The patient was followed at regular intervals. The last visit was one year after surgery at which time he was entirely free of evidence of recurrence, was in excellent health, and growing normally (Fig. 5).

Final Pathologic Diagnosis.—Following reconsideration of the pathologic specimen, the diagnosis originally entertained was replaced by that of "postinflammatory tumor of lung and anterior mediastinum."

DISCUSSION

Postinflammatory tumors of the lung as previously described, are characteristically solitary, circumscribed, and of uniform density varying from 2.5 to 7 cm. in diameter.[1, 2] They have been located in the parenchyma of the lung in the majority of cases but have been found in the pleura,[3] mediastinum,[2, 4] or been present in all 3 locations as in our case. Previous recurrent or concurrent lower respiratory infections have been present in most patients. Acute febrile pulmonary infections progressing concomitantly with such tumor formations are relatively rare. Umiker and Iverson describe but 2 of their 7 cases as occurring in this manner. Other cases have had free intervals of from 2 months to 22 years before the pulmonary lesions were found and identified.

The age distribution has varied from 3 to 61 years.[2, 5]

Evidence for the benignity of these pseudotumors has been their failure to recur. The youngest patient has lived 8 years without reappearance of the tumor.

In seems probable that an unresolving pulmonary or mediastinal density or one enlarging, as in our case, immediately following an acute lower respiratory infection, especially in the younger age group,[6] is an important clinical feature and should lead one to suspect a postinflammatory tumor.

The histologic features of these lesions include a proliferation of fibroblasts with or without production of conspicuous collagen, formation of numerous small vascular channels, and an admixture of inflammatory cells, chiefly plasma cells. Variable degrees of hemorrhage may occur and foam cells may become prominent. In reported cases there is a conspicuous lack of mitotic figures.

Because of the pleomorphic pattern and the quantitative variability of the components, various diagnoses have been applied to these lesions. They have frequently been mistaken for sarcomas.[1, 7] Diagnoses of fibroma, fibrous xanthoma or xanthoma have been entertained when the appropriate components predominate.[8, 9] It would appear that the quantity of xanthomatous elements are more closely related to the degree of hemorrhage than other factors, thus producing a tissue response similar to that observed in a dermatofibroma of the cutis. When plasma cells are a feature, the differentiation from a plasma cell neoplasm may be difficult. Many of the plasma cell granulomas of the lung and mediastinum as reported in the literature would appear to be

included in the subject under discussion.[2, 4, 5, 10] Some of these have involved lymph nodes as well as pulmonary tissue with partial or complete replacement of the nodal architecture. They may contain abnormal and juvenile forms of plasma cells indistinguishable per se from cells of multiple myeloma.[11] The relationship of the plasma cell granulomas of the mediastinum and lung to plasma cell lesions of the upper respiratory tract is still not completely settled. The true plasma cell neoplasms consist of a solid growth of plasma cells without inflammatory phenomena of vascularity, collagen production, and infiltration of varieties of other inflammatory cells.[12] The presence of Russell bodies in inflammatory lesions and their absence in neoplastic lesions has not proved a reliable criterion of differentiation.[11] None of the plasma cell granulomas presenting primarily in the lung or mediastinum have developed multiple osseous lesions of multiple myeloma.

SUMMARY

A clinical case of a 3½-year-old white boy is reported with the diagnosis of postinflammatory tumor of the lung and mediastinum. The gross appearance and extension of the process in the upper lobe of the right lung to the anterior mediastinum, in our opinion, gave ample justification for the extirpative management of the poorly delineated "tumor" which was initially diagnosed pathologically as being a malignant thymoma. Postoperative deep x-ray therapy, approximating that amount usually given in the treatment of radiosensitive malignant neoplasms of the lung and mediastinum, was given prior to arriving at the apparently correct pathologic diagnosis. (Irradiation therapy is not recommended for such "pseudotumors".) The patient is growing normally and is asymptomatic without roentgenographic evidence of residual pulmonary, pleural, or mediastinal involvement over one year after surgical removal of this uncommon and poorly understood chronic inflammatory lesion.

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SOLITARY BRONCHIAL POLYPS OF INFLAMMATORY ORIGIN

A Report of Two Cases Treated by Operation

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POLYPS are commonly found in the nose, the larynx, and the trachea. A solitary polyp in the bronchus is, however, very rare. Bednář,[2] Fourestier and associates,[4] and Šálek and co-workers[9] reported isolated cases and Lecoeur (cit. Fourestier) emphasized that in some thousands of bronchoscopies he had met with only one case. This low rate of incidence is confirmed in our clinic where, during the last 10 years, we have had only 2 cases of solitary bronchial polyp.

CASE REPORTS

Case 1.—V. J., a 61-year-old worker, stated that he had always been healthy, but that 10 years previously an irritative cough had developed with a small quantity of mucous expectoration. X-ray examination carried out 5 years later did not show any significant abnormality in the lung. About 3 years before admission, he developed pain in the back and the x-ray examination at that time showed a chronic bronchitis, emphysema of the lungs, and pleurisy with a small effusion in the right costophrenic angle. He had recently been losing weight. No hemoptysis had occurred.

X-ray examination on admission in 1952 showed an opacity in the right supradiaphragmatic field due to atelectasis of the middle and lower lobes of the right lung.

Bronchoscopy revealed in the bronchus intermedius a smooth, globular, pink, soft, easily movable tumor, the size and shape of a small cherry. A biopsy was taken and showed on histologic examination, only fragments of bronchial mucous membrane and inflammatory exudate.

A right thoracotomy on July 18, 1952, disclosed atelectasis of the middle and lower lobes with numerous adhesions to the parietal pleura. In the hilus, soft, moderately enlarged lymph nodes were found. The middle and lower lobes were resected and, when the bronchus intermedius was incised, a pink, lobular, soft tumor, the size of a cherry, protruded from its lumen.

The immediate postoperative course was uneventful and on review, 4 years after operation, he appeared to be perfectly well.

The examination of the specimen revealed a mass, 2 by 1.5 by 1.2 cm., inside the bronchus intermedius where it was attached by a relatively large pedicle to the inner wall of the bronchus close to its origin and nearly obliterating its lumen. The surface of the tumor was papillary; some of the papillae were rugged, cauliflower-like, while others showed smooth and globular form (Fig. 1). The tissue of the middle and lower lobes was the seat of chronic nonspecific pneumonia and was partly necrotic.

Histologically the tumor had a venous and fibrous stroma with some elastic fibers. In its central portion, deposits of fatty tissue were found (Fig. 2). The stroma was permeated by lymphocytes and plasma cells, especially near the surface of the papillae. The

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Received for publication Nov. 5, 1957.

surface of the tumor was covered with ciliated columnar epithelium (Fig. 3). The wall of the bronchus to which the tumor was attached was diffusely affected by chronic inflammatory changes.

The bronchial glands were distinctly enlarged.

The histologic findings led to the conclusion that this was a case of extreme hyperplasia of bronchial mucous membrane of inflammatory origin.

CASE 2.—In A. F., a 36-year-old blacksmith, there developed, 5 years before admission, a dry cough with some mucous expectoration, followed in 1952 by dyspnea and pain in the left chest. X-ray examination in 1954 revealed only a slightly increased opacity of the left lung. Bronchoscopy, carried out in a sanatorium in 1955 demonstrated a tumor in the left main bronchus. A fibroma or neurofibroma of the bronchus was suspected. With this diagnosis the patient was admitted to the hospital. In 8 or 9 months prior to admission he had lost 8 Kg. in weight. There had been no hemoptysis.

X-ray examination, tomography, and bronchography revealed a slight diffuse opacity of the whole of the left lung, caused by a partial obstruction of the main bronchus by a tumor within the bronchus.

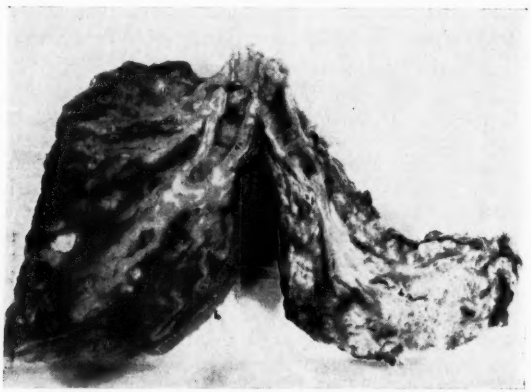


Fig. 1.—Case 1. The middle and lower lobe of the right lung. The lumen of resected bronchus is closed by the inflammatory bronchial polyp.

Bronchoscopy revealed in the left main bronchus, about 2 cm. below the carina, a smooth, globular, pedunculated tumor, the size of a cherry, attached to the posterior wall of the bronchus. Macroscopically, it appeared to be a lipoma.

Histologic examination of the bronchoscopic specimen, consisting of a part of the hypertrophic bronchial mucosa (Fig. 4), showed that to a large extent it was covered on the surface with proliferating, evidently metaplastic squamous epithelium. The lamina propria of the mucosa was very thick and edematous, containing some isolated, foamlike macrophages. In some places the interstitial tissue gave a slightly positive histochemical reaction for mucus. The histologic findings in the bronchoscopic specimen suggested an inflammatory polyp originating in bronchial mucosa with myxoid degeneration.

At operation on July 7, 1955, a slight diminution in aeration of the left lung was found. In the left main bronchus, a soft tumor was palpable. Bronchotomy was carried out and an oblong, pink, lobular, well-circumscribed tumor was found, which was attached to the wall of the bronchus about 1 cm. above the origin of the left upper lobe bronchus by a slender pedicle about 3 mm. in diameter. The tumor nearly closed the lumen of the main bronchus and extended into the upper lobe bronchus. The pedicle was excised together with its bed, the mucus was aspirated from the bronchial tree, and the bronchus sutured. The lung re-expanded very well.

Fig. 2.

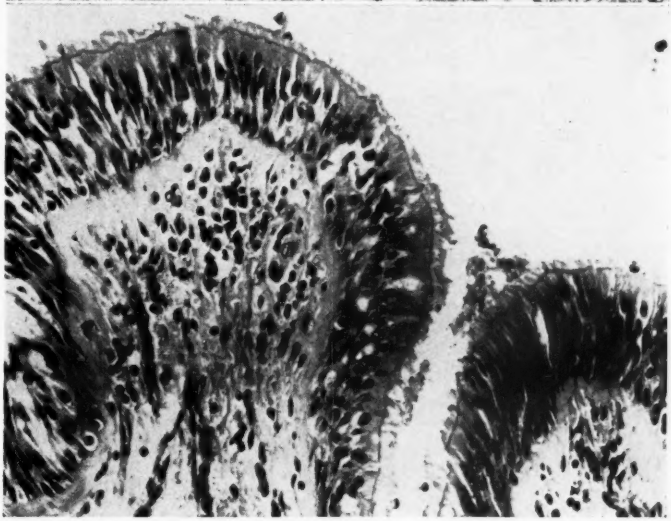


Fig. 3.

Fig. 2.—Case 1. The pedicle of the bronchial polyp (hematoxylin-eosin-elastic; ×22).
Fig. 3.—Case 1. The surface of polyp covered with stratified ciliated columnar epithelium (hematoxylin-eosin; ×290).

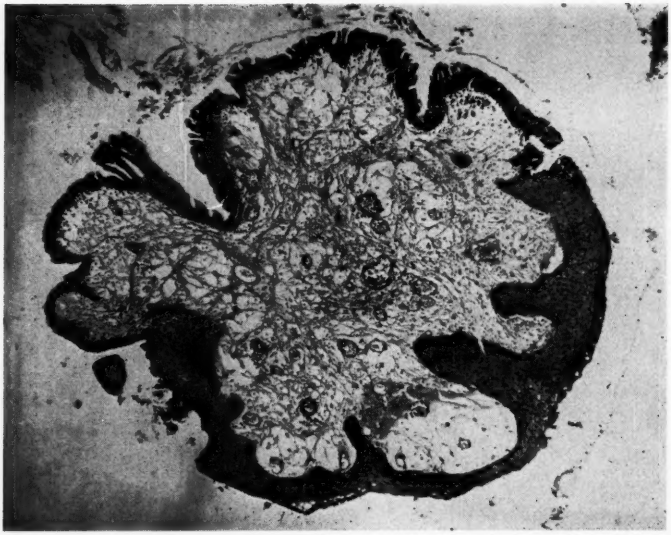


Fig. 4.—Case 2. The fragment of bronchial mucosa removed at bronchoscopy (hematoxylin-eosin; ×40).

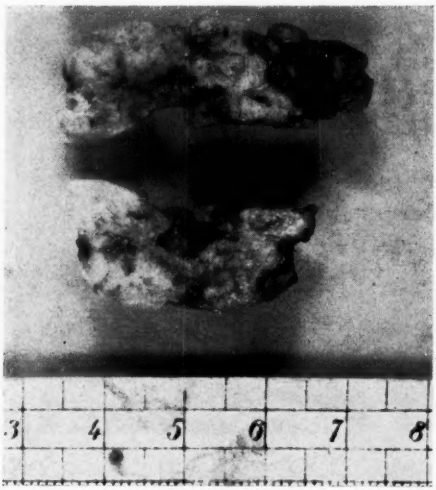
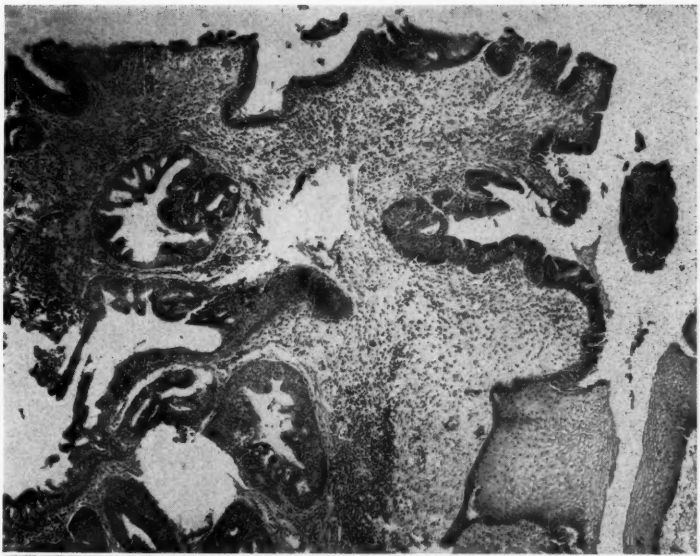


Fig. 5.—Case 2. The dissected polypus removed by bronchotomy.

Fig. 6.



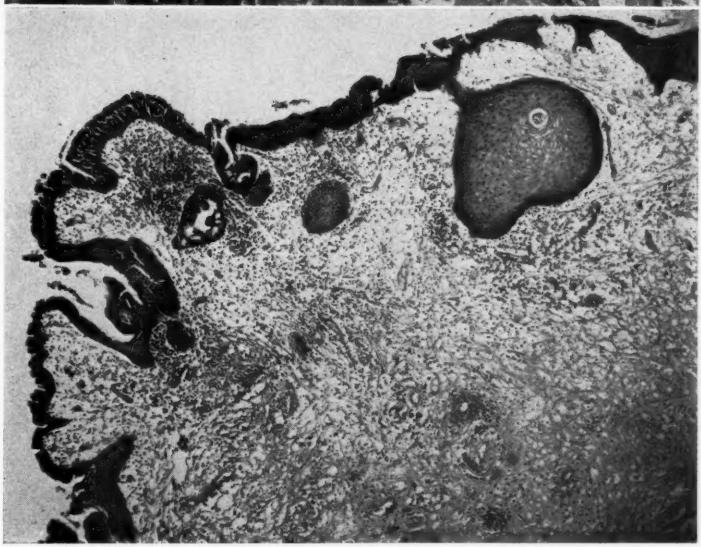


Fig. 7.

Fig. 6.—Case 2. The bronchial polypus is covered with columnar and squamous epithelium (mucicarmine; ×50).

Fig. 7.—Case 2. The columnar and squamous epithelium on the surface of polypus (hematoxylin-eosin; ×50).

Fig. 8.

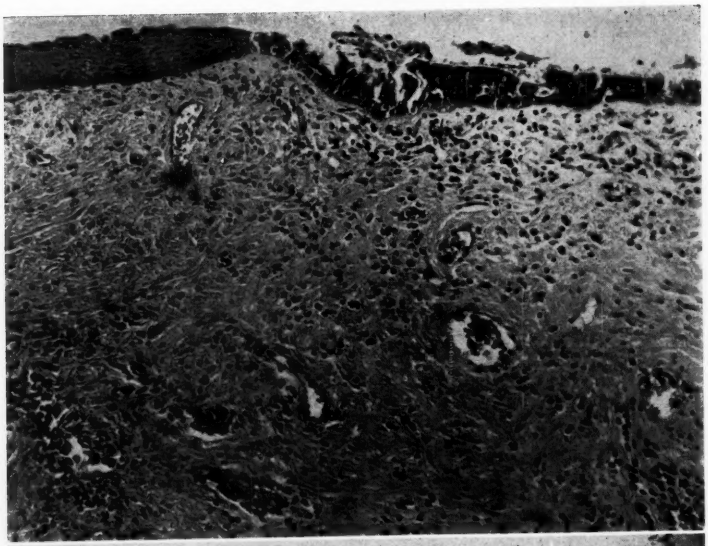


Fig. 9.

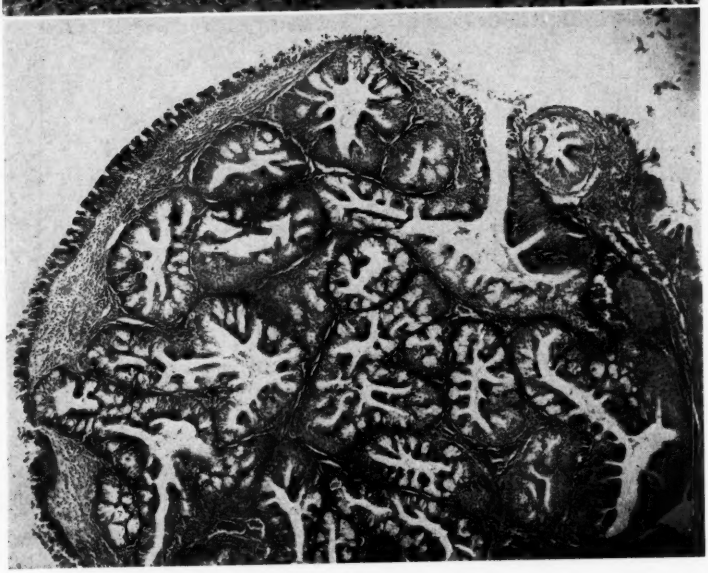


Fig. 8.—Case 2. A round-celled inflammatory infiltration of the stroma (hematoxylin-eosin; ×115).

Fig. 9.—Case 2. Some details from the polypus simulating the glandular structures (van Gieson; ×40).

The patient recovered and a subsequent bronchoscopy demonstrated adequate patency of the main bronchus; there was no recurrence of the tumor.

The tumor measured 40 by 11 by 12 mm. (Fig. 5) and was of a grayish-white color, with a rough surface, and of soft consistency with pink-red deposits on the cut surface.

Microscopic examination revealed a sparse, fibrous stroma which on the surface was covered partly by ciliated columnar epithelium (Fig. 6), but mostly by metaplastic, stratified, squamous epithelium (Fig. 7). Some parts of the stroma contained large numbers of fine collagenous fibers with small groups of siderophages. Around the dilated veins there was a round-celled inflammatory infiltration (Fig. 8). Some sections were very edematous and, in places, signs of myxoid changes were evident. The covering epithelium contained numerous goblet mucus cells. The epithelium was marked by a striking proliferation in the form of solid columns of stratified squamous cells or in the form of closely connected papillary or cystopapillary formations (Fig. 9). Although the proliferation of the epithelium was considerable, no abnormal cell types or atypical mitoses were found. The process had no malignant character.

In the lymph glands there was anthracosis and chronic nonspecific inflammatory reaction.

Histologically, it appeared to be a case of a circumscribed polypoid hypertrophy of bronchial mucosa with squamous metaplasia caused most probably by a chronic inflammation.

DISCUSSION

Bronchial polyps because of their rarity seldom come to the attention of the clinician and then not until their secondary effects are evident. More frequently, they are seen by the pathologist at necropsy as a variant of the slight polypoidal hypertrophy of the mucosa which macroscopically need not look like a polyp. The histologic structure of both forms is, however, analogous (Bednář[2]). The polyps are usually located in the main bronchus or its principal branches, either close to their origin or directly at the bifurcation of the bronchus. Lecoeur maintains that this location is characteristic for inflammatory polyps.

Clinically large polyps manifest themselves in the same manner as benign tumors of the bronchus. Their symptomatology depends in many respects not only on the size of the polyp and on the diameter of the bronchus, but also on the character of the underlying inflammatory process. Jackson and Jackson[7] contend that two symptoms are always present, hemoptysis and wheezy breathing, the latter particularly when the large bronchi are affected. Neither of our 2 patients presented these symptoms, but each had had a cough for 10 and 5 years, respectively; no doubt a manifestation of chronic inflammatory changes in the bronchial mucosa which were seen in Case 1. From the histology we could not, however, ascertain the cause of this bronchitis. Clinical symptoms attributable to the polyp manifested themselves in these 2 cases much later than the symptoms of bronchitis, 7 and 2 years, respectively, from the onset of persistent cough. These clinical features, however, are in no way pathognomonic, for correct diagnosis can be made only by bronchoscopic biopsy.

Macroscopically, these polyps are circumscribed masses of a globular or oval shape, attached by a pedicle to the inner wall of the bronchus. Their surface is smooth but lobular, sometimes velvety or even rough and, in isolated cases,

ulcerated (Jackson[7]). Their color as seen by us at bronchoscopy and at operation is light pink or reddish and the polyp is frequently veiled by a fine covering of mucus. They are relatively friable.

The question remains whether polyps should be classed as real tumors or as examples of inflammatory hyperplasia. Histologic structure of the polypus, particularly when it is marked by papillomatous arrangement which we have seen in both the above cases, makes it necessary to consider a neoplastic process both histogenetically and in the differential diagnosis. In support of this possibility is the fact that, in some cases, the inflammatory cellular infiltration of the stroma is slight and sometimes even the squamous metaplasia of the covering epithelium is not present (Case 1). Therefore, some authors (Jackson,[7] Hollmann,[6] Lindgren[8]) classify these polyps as benign bronchial tumors, perhaps analogous to fibro-epithelial neoplasm in other locations, as for instance condyloma acuminatum (Bednář), cutaneous papilloma, uterine mucous polyps, etc. The nature of these conditions is, however, also doubtful.

The relation of bronchial polyps to diffuse segmentary hypertrophy of the bronchial mucosa, which was described by Horányi[5] under the term "bronchial adenosis," is not yet clear. In about 3 per cent of 300 resected lungs, Horányi found circular stenosis of the bronchial lumen caused by hypertrophy of the mucosa with a striking increase and cystic enlargement of bronchial glands. He characterizes the process as a non-neoplasmatic hyperplasia but contests its inflammatory origin. A similar observation was made by Eszipova[3] in one case. We assume that inflammatory polyps can be a component part, though rarely, of chronic hypertrophic bronchitis. The inflammatory nature of this process is also admitted by Fourestier[4] and Bariety and his associates.[1]

We are of the opinion that polypoid hypertrophy of the bronchial mucosa is caused by a similar process as is operative in the development of mucous polyps in the nose. An inflammatory process, even localized, can produce a secondary narrowing of the lymphatic vessels and thereby a condensation of tissue fluid in the affected part of the polypoid hypertrophic mucosa. That is also the causative process in circumscribed edema of the mucosa, according to the observations of Bariety. Such an explanation would be sustained especially by the histologic appearance of the above reported Case 2. The question of the infective agent which brings about this inflammatory process remains unsolved. It seems that any inflammatory condition in the bronchus accompanied by a stagnation of the secretion, particularly when purulent, can be the decisive factor.

SUMMARY

Two cases of solitary inflammatory bronchial polyps which clinically manifested themselves by a cough of long duration and by later obstruction of the bronchi are reported. Both patients were treated by operation, one by lobectomy and the other by bronchotomy.

The authors discuss the genesis of these polyps and express the opinion that they are caused by chronic inflammation of the bronchial mucosa.

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HAMARTOMA OF THE LUNG

Report of One Malignant and Three Benign Cases

Elwyn Cavin, M.D.,* Joseph H. Masters, Major, MC, USA, and James Moody, M.D.**

ALTHOUGH the number of reported cases of pulmonary hamartoma is steadily increasing, there are few instances of reported enlargement of the tumor while the patient is under observation. The presence of malignant changes in these tumors is likewise rare. A case of a rapidly enlarging malignant hamartoma in the left lower pulmonary field of an infant, 22 months old, is the primary reason for this report. Three additional cases of benign pulmonary hamartoma are also included. These 4 cases were discovered at the center for Thoracic Surgery established by the United States Army Medical Service in Europe, between March, 1954, and March, 1956.

CASE REPORTS

Case 1.—A baby, 22 months old, was first seen in August, 1954, because of a nodular shadow in the lower left pulmonary field discovered in a routine roentgenogram of the chest (Fig. 1). The patient had no thoracic symptoms and a decision was made to have her return later for further study. When the child returned in March, 1955, she still had no thoracic symptoms, and the only positive physical findings were increased breath sounds and dullness in the left lower pulmonary field, and definite Mongoloid features and retarded development.

Considerable increase in the size of the lesion was noted in roentgenograms of the chest; in fact the entire lower half of the left pulmonary field was obscured by the tumor (Fig. 2, *A*). The upper border of the mass was convex and smooth in outline. The heart and mediastinum were displaced to the right. In the lateral view the mass could be seen lying posteriorly with the anterior border reaching to the mid-portion of the cardiac shadow (Fig. 2, *B*). At fluoroscopy, the mass moved freely with respiration and could not be separated from the diaphragm. This led the observers to conclude that the lesion originated not from the mediastinum but rather from pulmonary tissue, and was possibly an enlarging bronchogenic cyst or teratoma.

Left lower lobectomy resulted in removal of the entire tumor. Recovery was uneventful and the patient was discharged in April, 1955. In March, 1957, there was no evidence of recurrence of the tumor.

The specimen was a lobe of lung containing a well-defined and circumscribed, nonencapsulated, fleshy, creamy white, trabeculated, solid tumor, 8 by 8 by 5 cm., with over two thirds of its surface covered by a thin pleura and the remainder of its margin attached to the adjacent compressed pulmonary parenchyma. The pleural surface was smooth and portions of the pleura could be stripped from the tumor. Where the tumor was adjacent

Received for publication Jan. 6, 1958.

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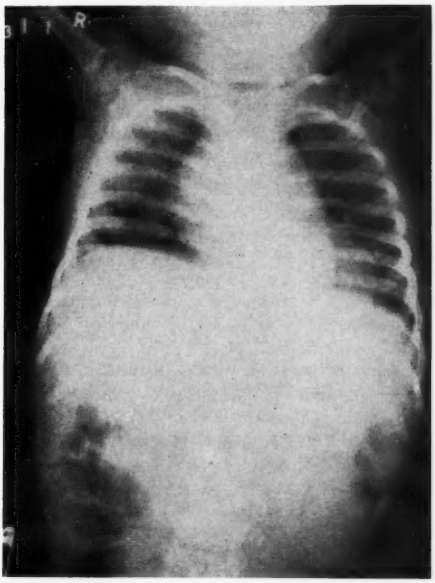
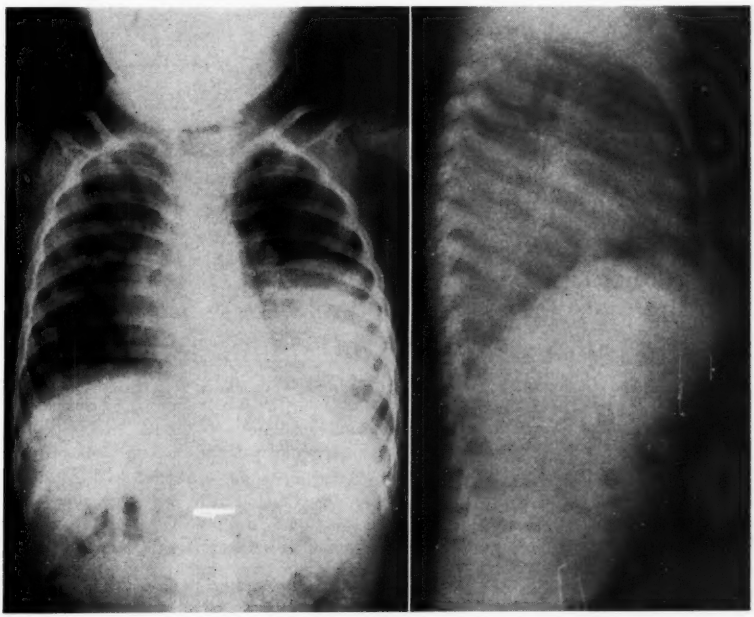


Fig. 1.—Case 1. Mass in lower left pulmonary field partially obscured by left cardiac border.



A. *B.*

Fig. 2.—Case 1. *A*, Film 7 months later showing pronounced increase in size and density of mass.

B, Lateral view showing posterior location with anterior border reaching to mid-portion of cardiac shadow.

to pulmonary parenchyma, the tumor and lung were not separated by a capsule. On cutting through the tumor, the pathologist felt a fine gritty sensation and there were small, white, semitranslucent foci in the medial portion of the tumor. This appeared to be cartilage.

Frozen sections revealed cellular mesenchymal tissue considered to be fibrous or smooth muscle, and the lesion was considered benign, probably representing fibroma or hamartoma of the lung. Microscopic examination of sections from the medial portion of the tumor revealed areas containing hyaline cartilage, fibrous tissue, bronchi, and smooth muscle tissue in a disorderly arrangement. Other sections were composed almost entirely of richly cellular intertwining tissue bundles which, on differential staining, were identified as smooth muscle tissue. It was stated that recent proliferation of the smooth muscle component of the tumor probably accounted for the recent growth observed in the roentgenograms. The tumor was considered to be benign and one lymph node in the specimen showed no neoplastic tissue.

The case was subsequently reviewed by several members of the Armed Forces Institute of Pathology who voiced the unanimous opinion that "the abnormal amount, distribution and morphology of the cartilage component of the lesion frequently related to clefts lined by normal-appearing bronchial epithelium suggested that the lesion was basically a hamartoma. However, the predominating spindle cells, arranged for the most part in interlacing bands, could not be interpreted as simply a component of a hamartoma. The spindle cell

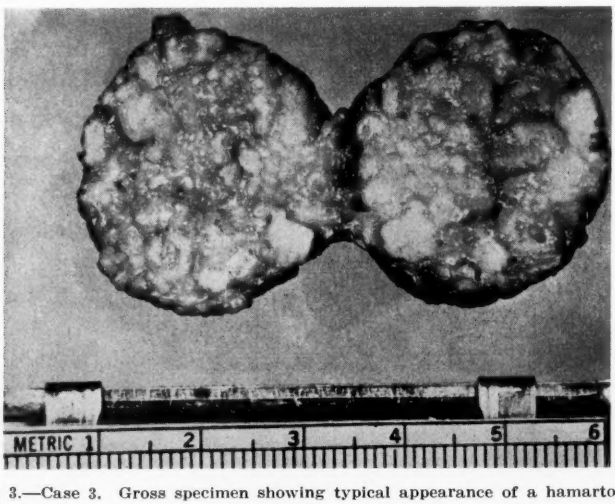


Fig. 3.—Case 3. Gross specimen showing typical appearance of a hamartoma.

growth was believed to represent a true neoplasm, probably of smooth muscle fibers and probably malignant because of the nuclear hyperchromatism and considerable mitotic activity. The lesion as a whole was, therefore, interpreted as a hamartoma in which the smooth muscle component had undergone neoplastic change. The case was coded as lobe of lung, probably leiomyosarcoma arising in hamartoma of lung. The reviewer was not aware of any instance of leiomyoma or leiomyosarcoma arising in this way."

CASE 2.—A white woman, 27 years old, was found to have a nodule, 2 cm. in diameter, in the right lower pulmonary field adjacent to the subhilar region. The tumor had smooth margins and its density was only slightly greater than that of the surrounding normal pulmonary tissue. The patient was asymptomatic. Results of tuberculin and coccidioidin tests were negative. The small tumor was enucleated with ease from the right upper lobe and the patient's recovery was uneventful.

Microscopic examination revealed a complex picture of multiple papillary processes and cystic spaces set in a stroma with a thin and indefinite capsule surrounding the whole mass. Many areas of fatty degeneration were present and in a few places the structure suggested cartilage of a primitive type. The lesion was considered to be a hamartoma but its superficial resemblance to a mixed tumor was noted.

CASE 3.—A white man, 35 years old, was found to have a nodule in the left pulmonary field on routine roentgenography of the chest on admission to the hospital. He had no thoracic symptoms. In the lateral view the tumefaction was located in the area corresponding to the lingula. Definite calcification could not be demonstrated in planigrams. At thoracotomy, the nodule was easily shelled out and the patient made an uneventful recovery. Cross section of the gross specimen showed the nodular, glistening appearance of the mass when freshly cut (Fig. 3).

CASE 4.—A white man, 47 years old, had evidence of a nodule, 1 cm. in diameter, in the right upper pulmonary field on routine roentgenography of the chest. Since no calcium could be identified within the lesion, removal was advised. The excised tumor, measuring 1.0 by 1.2 cm., was rounded, slightly lobulated, white, and firm. Sections showed a disorderly arrangement of cartilage, fat, fibrous tissue, and myxoid stroma. In this matrix were numerous, poorly formed bronchi, many of which showed a papillary configuration.

DISCUSSION

Albrecht[1] is generally credited with originating the term hamartoma in 1904. Since then, tumors containing an abnormal mixing or arrangement of the normal components of an organ have usually been designated as hamartomas. However, in the past, many tumors that were found in the pulmonary fields and were called chondromas, enchondromas, lipochondromas, lipo-osteoadenochondromas, or whatever tissue combination existed in disorderly arrangement, were undoubtedly hamartomas. McDonald and associates[2] pointed out that many tumors reported as chondromas in the older literature were actually hamartomas.

At times the arrangement of the tissue components is such that classification is difficult. Two of the cases presented by Lemon and Good[3] were atypical in histologic appearance. Freedlander and associates[4] reported a case in which there was disagreement among the pathologists concerning the diagnosis of a tumor removed from the pulmonary fields of a woman 56 years old; they believed that this tumor, which incidentally had increased in size on successive examinations, was a hamartoma. In 3 exceptional cases, reported by McDonald and associates,[2] the tumor did not contain cartilage.

There are several interesting features in Case 1. The rapid growth of the tumor over a period of 7 months is unusual. Weisel and co-authors[5] reported 10 hamartomas, all of which had shown evidence of growth on serial films. Most of these had increased in size from a few millimeters to 2.5 cm. over periods of observation of 2 years. In one of the cases cited by Stein and Poppel[6] there was, during a period of 12 years, a slight increase in size which could not be completely accounted for on technical factors. However, most of the reported cases are similar to the 2 cases reported by Hall[7] followed 5 and 21 years, respectively, by repeated roentgenographic examinations with no demonstrable increase in size.

The malignant changes present in the tumor in Case 1 as interpreted by members of the Armed Forces Institute of Pathology prompted search of the literature for similar cases. Sherwood and Sherwood[8] reported a fatal case of chondroma of the lung. Simon and Ballon[9] described local cancerous features in a case of hamartoma of the lung.

Also of interest is the young age of the patient in Case 1. In a review of 107 cases of chondromatous hamartomas by Jones,[10] in 1949, the ages ranged from 21 to 78 years. Jones reported a hamartoma, found at necropsy in a newborn infant, which occupied almost the entire upper lobe of the lung.

In none of the 4 cases presented here was calcium demonstrated roentgenographically. Although several authors have emphasized the frequency of calcification in these tumors, it apparently is not as frequent a finding as we have been led to believe. In the 17 cases presented by Lemon and Good,[3] only 3 contained calcium.

There were no roentgenographic features in these 4 cases that would have suggested the diagnosis of hamartoma with a reasonable degree of accuracy. Two of the nodules were opaque to the roentgen rays whereas two were relatively radiolucent with density only slightly greater than that of the surrounding pulmonary tissue. This radiolucency can be ascribed to the high fat content and cystic spaces within the nodular mass. The subpleural location and well-defined borders without reaction of the adjacent pulmonary tissue were features noted that have been previously reported. The latter would assist in excluding tuberculoma, but there are a host of benign lesions and a few malignant ones that have fairly clear-cut borders.

SUMMARY

Four cases of hamartoma of the lung have been presented. One of these tumors, in a baby 22 months old, increased greatly in size over a period of 7 months and microscopic examination revealed malignant changes in the smooth muscle component of the lesion.

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INEFFECTIVENESS OF ADRENOSEM IN PULMONARY SURGERY

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ADRENOSEM (adrenochrome monosemicarbazone sodium salicylate complex) is a substance related chemically to epinephrine, but it is devoid of sympathomimetic effects. Its use as a hemostatic agent was suggested by the studies of Roskam,[1] who showed that epinephrine favors spontaneous hemostasis after it is oxidized into adrenochrome or a closely related substance. Additional studies by Fulton and co-workers,[2, 3] on the cheek pouch of the hamster, indicated that petechial formation which usually follows tissue trauma is reduced by Adrenosem. It is claimed by the manufacturer that the compound decreases capillary permeability and enhances retraction of severed capillary ends. A number of recent reports concerning the use of the drug to control small vessel bleeding during surgery have been enthusiastic. Peele[4, 5] reported favorable results in tonsil and adenoid operations. He felt that bleeding was diminished by the use of Adrenosem, as evidenced by reduced operating time and less requirement for suture. Cases of epistaxis had decreased need for postnasal packing; and the operative field was better visualized in subglottic surgery. Owings[6] claimed a reduction in postoperative bleeding from 10 per cent to 2 per cent with Adrenosem therapy in nose and throat work. Sherber,[7] Riddle,[8] and Bacala[9] have described their experiences with the drug in a number of surgical procedures, the results of which were all favorable. Some skepticism was expressed, however, in a recent A.M.A. report.[10]

It is difficult to evaluate these reports because of their subjective nature. To date, no studies have been described in which adequate control data were provided. In the past, many alleged hemostatic agents have received brief periods of enthusiasm and were subsequently shown to be ineffective. Perhaps few other areas are so difficult to intepret because of spontaneous variation and lack of strict criteria for effect. The present study was undertaken to attempt an objective evaluation of Adrenosem as a hemostatic agent during surgery.

METHOD

The subjects were patients undergoing thoracic surgery at Montefiore Hospital. In all cases, pneumonectomy or lobectomy was the anticipated operation. The relatively uniform procedure performed by the same operating team appeared to represent a situation most favorable for evaluation of a hemostatic

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Received for publication Oct. 9, 1957.
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agent. Moreover, this type of operation involved considerable bleeding from small vessels. One half received intramuscular Adrenosem,* the other half received injections of a placebo of similar appearance. During the study, the ampules were labeled only by code letters. The key to the code was not available to this group until after the series was completed, and the surgeons did not know which type of injection was given. The two types of ampules were given in a random and predetermined order. Dosage schedules were according to the manufacturer's instructions.

A few days before surgery each patient had a one-stage prothrombin test and a platelet count, performed by phase microscopy. In patients who came to surgery, these were within normal limits. One hour before operation, a Rumpel-Leede test and bleeding time were performed. The Rumpel-Leede test was done with the blood pressure cuff inflated to 90 mm. Hg for exactly 5 minutes. The bleeding time was performed by piercing the ear lobe completely with a No. 11 Bard-Parker blade. At this time 10 mg. (2 ampules) of "medication" were given. Just prior to anesthesia, the Rumpel-Leede test and bleeding time were repeated. Subsequently, intramuscular injection of 5 mg. was given by the anesthetist (a) when the chest was opened, (b) when the operation was deemed half over, as estimated by the surgeon. Additional 5 mg. injections were given 2 and 5 hours postoperatively.

The patients were closely observed for the degree of bleeding, especially small blood vessel oozing. Bleeding from major vessels did not influence the ratings. The postoperative course was evaluated for evidence of further blood loss into the pleural cavity and the operative wound. A final evaluation was made of bleeding during the entire procedure, including the postoperative course, and was classified as more, the same, or less bleeding than expected for that particular type of operation.

TABLE I. RESULTS OF RUMPEL-LEEDE TEST

RESULT	PLACEBO	ADRENOSEM
0	23	21
1+	1	4
2+	1	0
3+	0	0
4+	0	0

*Provided by the S. E. Massengill Company, Bristol, Tenn.

RESULTS

Table I shows the result of the Rumpel-Leede test performed one hour before surgery, at which time the first dose of "medication" was given. The same test was repeated immediately before surgery. In no cases was there a change. Table II indicates that there were only minor changes in bleeding time in the one-hour interval. Total evaluation of the results is shown in Table III. It appears that there is no demonstrable difference in the amount of bleeding and oozing when a placebo is used in place of Adrenosem.

TABLE II. CHANGE IN BLEEDING TIME DURING ONE-HOUR INTERVAL BEFORE SURGERY

CHANGE (MIN.)	PLACEBO	ADRENOSEM
+1	1	3
No change	21	20
−1	3	2

TABLE III. TOTAL EVALUATION OF BLEEDING AND OOZING

	MORE	SAME	LESS
Adrenosem	7	6	12
Placebo	6	9	10

DISCUSSION

The data indicate that Adrenosem has no effect in reducing small blood vessel bleeding in pulmonary surgery. Although this type of operation may present special problems, hemostasis is tested in normal skin, muscle, and other tissue. The burden of proof, therefore, is on the shoulders of those who claim that the drug does have benefit. Existing concepts of hemostasis in small blood vessels[11, 12] indicate that the most important single factor involved is the blood platelet. When small vessels are cut, agglutination of the platelets and their adhesion to the damaged vascular wall is the primary event. (In those vessels large enough to have a muscular coat, vasoconstriction takes place first.) Then follows the mechanism of blood coagulation, with reinforcement of the platelet plug by a fibrin clot. A hemostatic drug, to be effective, should have one or more of the following properties: (a) improve vascular resistance, (b) aid in the formation of the platelet plug, and (c) increase blood coagulability. These effects have not been noted with Adrenosem.

Casual clinical observation cannot be regarded as a reliable method of drug evaluation. Adequately controlled study, preferably using a double blind technique, gives the objective information needed before definite conclusions can be drawn. The situation is reminiscent of periodic discoveries of drugs to treat hemophilia. Such agents as estrogens and ergot alkaloids were stated to be the treatment of choice for this condition but, when results were carefully examined by objective methods, the initial enthusiasm could not be confirmed.[13]

SUMMARY AND CONCLUSIONS

The drug Adrenosem was studied, using a double blind technique. There was no appreciable difference between the effect of the drug and a placebo in the course of its use in pulmonary surgery. We conclude that Adrenosem probably is of no value as a hemostatic agent.

We wish to acknowledge the cooperation of Dr. Elliot Hurwitt, Dr. Morton Rosenblatt, Dr. Aaron Schwartz, and Dr. Stanley Fell, the surgeons whose participation made this study possible.

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THE AMNESIC EFFECT OF PROMETHAZINE HYDROCHLORIDE IN BRONCHOSCOPY

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AMNESIA is a desirable phenomenon in the patient undergoing bronchoscopic examination. Bronchoscopy under topical anesthesia in the conscious or alert patient is an unpleasant experience for both patient and endoscopist. For adequate examination of patients who cannot cooperate, bronchoscopies are frequently performed under general anesthesia. However, the bronchoscope cannot be introduced in the somnolent patient unless the jaw muscles are relaxed.

Relaxation can be achieved by use of general anesthesia, by other central nervous system depressants, or by a muscle relaxant such as succinylcholine chloride. However, these techniques tend to produce respiratory depression. Hypoxemia can result, with all its grave consequences. To avoid hypoxia and hypoxemia, a portable chest respirator has been employed in conjunction with anesthesia for bronchoscopy.[1]

In our opinion, general anesthesia or use of the chest respirator plus topical anesthesia introduce unnecessarily complicating factors into bronchoscopy. Such procedures inevitably involve more risk to the patient than actual insertion of the bronchoscope.

Ideally, bronchoscopy calls for: (1) an awake, cooperative patient who can respond to and obey requests of the operator; (2) a relaxed and comfortable patient with a sense of well-being; (3) a patient without respiratory depression; and (4) one who later lacks unpleasant memory of the bronchoscopic procedure.

Anderson[2] performed 290 bronchoscopies with cocaine hydrochloride topical anesthesia and intravenous meperidine hydrochloride as a sedative-analgesic. His patients were reportedly comfortable and relaxed, although awake and cooperative. Some cases were amnesic.

Altman and Fialkov[3] reported 156 bronchoscopies performed with tetracaine hydrochloride-cocaine hydrochloride topical anesthesia and intravenous meperidine hydrochloride as a sedative-analgesic. No anxiety was noted during bronchoscopy and amnesia was observed in a few patients.

The use of promethazine hydrochloride (Phenergan hydrochloride) in bronchoscopic examination has not been reported, yet the drug would appear to hold merit for such procedures. Sadove,[4] Weiss and McGee,[5] and Holzmann and Josipowicz[6] described the sedative and antiemetic action of promethazine, and its value in anesthetic techniques. None of these investigators emphasized the amnesic effect of promethazine, although Weiss reported its existence in older patients.

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Received for publication Dec. 4, 1957.

PLAN OF STUDY

Bronchoscopic examination was performed on 50 consecutive, unselected patients where the procedure was indicated. Twenty-five patients received intravenous meperidine hydrochloride for sedation and analgesia, and hexylcaine hydrochloride for topical anesthesia. The remaining 25 cases received intravenous promethazine hydrochloride* in addition to meperidine and hexylcaine.

Method.—In general, preoperative medication consisted of: (1) pentobarbital or Secobarbital, 90 mg., by mouth 90 minutes prior to bronchoscopy, and (2) 60 mg. codeine hydrochloride and 0.43 mg. atropine sulfate in a single intramuscular injection 60 minutes before bronchoscopy.

At time of examination, the patient lay in supine position on the bronchoscopy table. An intravenous drip of 5 per cent dextrose in water was started. A three-way stopcock in the tubing facilitated control of the intravenous drug injections. Blood pressure, pulse rate, respiratory rate, and depth were monitored throughout examination.

Among those receiving promethazine, induction was begun with a single 50 mg. intravenous injection of the drug. Immediately thereafter, 20 mg. meperidine was introduced through the tubing. The patient was then questioned and observed for relaxation and a "feeling of lightheadedness." The meperidine dose was repeated in 20 mg. increments at 2-minute intervals until the patient appeared to sleep lightly.

In the "dozing" stage, patients who received promethazine responded to commands, were cooperative, and were easily aroused. To reach this stage of sedation, the mean meperidine dose was 130 mg. and ranged from 40 to 220 mg. At least 15 minutes were required for peak effect of the meperidine.

When the patient appeared relaxed, the posterior pharynx and larynx were sprayed with a 5 per cent aqueous solution of hexylcaine hydrochloride. After anesthetization of the vocal cords, 2 c.c. of 5 per cent hexylcaine solution were injected between them. A syringe and flexible laryngeal cannula facilitated this last operation which served to anesthetize the tracheobronchial tree. Bronchoscopy, was then undertaken.

The 25 cases not receiving promethazine were treated in identical manner, save that induction started with introduction of 20 mg. meperidine. Among these patients, the "dozing" state could not be attained without a meperidine dosage that would also bring about unwanted respiratory depression. Thus, bronchoscopy in these patients began when the patient was apparently well relaxed after a mean meperidine dose of 150 mg.

The sedative and anesthetic procedures involved an average of 20 minutes in all 50 patients. Amnesia or its lack was ascertained by close and individual questioning of each patient postoperatively. A standard set of questions were put which traced the patient's memory of events minute by minute from the time of leaving the ward. Complete amnesia was recorded only if the patient had no memory whatever of the procedure.

*Promethazine hydrochloride (N-[2'-dimethylamino-2'-methyl] ethyl phenothiazine hydrochloride) was supplied for this study by Wyeth Laboratories, Inc., Philadelphia, Pa., as Phenergan® hydrochloride.

RESULTS

Meperidine and Hexylcaine.—No patient experienced complete amnesia during examination. The typical patient partially recalled the procedure and remembered periods of stimulation by the bronchoscope. No patient felt the experience was unpleasant and all expressed willingness to undergo bronchoscopy again if necessary. All subjects were awake and oriented at the end of the procedure. They were returned to the wards immediately and needed little postoperative care. Only 2 of 25 patients attained the desired stage of relaxation on 100 mg. of meperidine or less. No laryngospasm was observed. Table I lists other pertinent data and tabulates results.

TABLE I. COMPARATIVE AMNESIC RESPONSE TO BRONCHOSCOPY FOLLOWING ANESTHESIA

ANESTHETIC TECHNIQUE	INTRAVENOUS MEPERIDINE HCl PLUS TOPICAL HEXYLCAINE HCl	INTRAVENOUS PROMETHAZINE HCl AND MEPERIDINE HCl PLUS TOPICAL HEXYLCAINE HCl
Number of patients	25	25
Age range, years	17-71	17-80
Median age, years	38	39
Range of meperidine HCl dose, mg.	90-200	40-220
Mean meperidine HCl dose, mg.	150	130
Promethazine HCl dose, mg.	--	50
Response		
Complete amnesia	0	15
Partial amnesia	1	6
No amnesia	24	4
	25	25

Promethazine, Meperidine, and Hexylcaine.—Of 25 patients, 15 had complete amnesia during bronchoscopy. Six vaguely recalled momentary and not unpleasant stimulation at some time during the procedure. Of those who remembered examination, none found it unpleasant. All were cooperative and obeyed commands when given. All patients were returned to their wards shortly after the procedure. Patients were awake or easily aroused and needed little postoperative care. Ten of 25 patients required 100 mg. of meperidine or less for adequate response. No laryngospasm was observed. Table I lists results.

With 3 patients, meperidine induction was intended. They became tense and uncooperative during an early stage of induction, and so 50 mg. of promethazine was given intravenously. All 3 then relaxed; induction was completed and the procedure was carried out without incident and with complete amnesia. These cases were tabulated as partially amnesic since memory was retained of the attempted meperidine induction.

Two patients experienced amnesia that began only 2 or 3 minutes after promethazine injection. In some cases the amnesic effect extended well into the postoperative period. Several patients commented they had "slept" up to 3 hours after bronchoscopy although in fact they had been easily aroused and responded to commands during this period.

Each of 2 patients underwent two bronchoscopies, one with promethazine and one without the drug. Both experienced complete amnesia when promethazine

was employed and no amnesia when the drug was not used. One of these 2 patients disliked the amnesic sensation under promethazine-meperidine anesthesia and preferred to retain his memory of events.

A careful watch was maintained for untoward response, particularly any change in blood pressure, pulse rate, and respiratory rate. No significant difference was observed in these physiologic phenomena and no side effects were noted.

COMMENT

Discomfort and pain associated with bronchoscopy accomplished solely under topical anesthesia leaves the patient with an unpleasant memory. Also, under such conditions, an adequate examination is not always possible.

Promethazine affords the patient amnesia and obliterates the memory of an unpleasant endoscopic procedure. Meperidine fortified with promethazine is an even more desirable drug regimen in that the sedative-analgesic combination provides a sense of well-being undisturbed by pressure or position discomfort. This procedure avoids use of general anesthesia and the attendant risks to obtain desired cooperation and relaxation. Table I also suggests promethazine permits use of less analgesic than would otherwise be required to carry out bronchoscopy without discomfort.

Our experience leads us to the opinion that amnesia is most desirable during bronchoscopy. Hence, we consider promethazine essential to the proper preparation of the patient for such procedures. The technique described here would appear suitable for wider usage, although local circumstances may call for slight modifications.

SUMMARY

Fifty patients underwent bronchoscopy; 25 received meperidine hydrochloride intravenously and topical anesthesia before examination; 25 received, in addition, promethazine hydrochloride intravenously. Of those receiving promethazine, 15 of 25 were completely amnesic and 6 more were partially so. No complete amnesia was observed among the 25 patients who did not receive promethazine. In our study, promethazine produced temporary amnesia and excellent relaxation without accompanying cardiovascular or respiratory depression or undesired side effects.

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RIGHT-LEFT IDENTIFICATION OF BRONCHOSCOPY SMEARS

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BRONCHIAL secretions, like respiratory air, share the upper respiratory tract to the right and left bronchi. Since this common passageway is both an entrance and an exit, there exists ample opportunity for mixing secretions on expiration and contralateral inoculation on inspiration. It is therefore impossible for the bronchoscopist to collect a smeared or aspirated specimen which represents a single side exclusively. Regardless of this limitation, determination of a preponderant bacterium or type of desquamated cell on a single side may aid greatly in diagnosis.

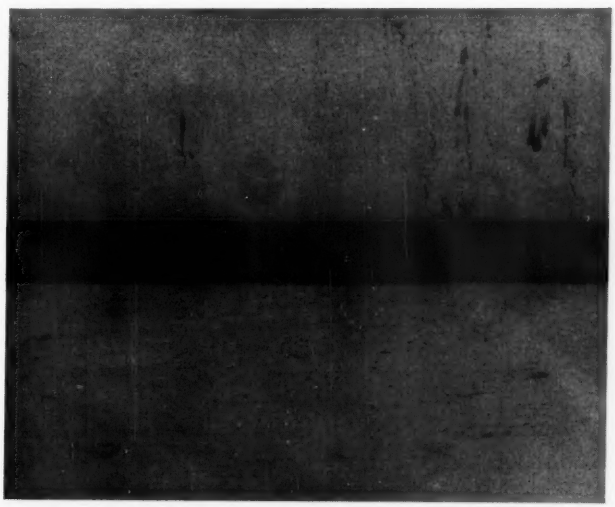

Fig. 1.—*Top*, Papanicolaou smear from right side. *Bottom*, Acid-fast smear from left side.

When nonlocalizing but symptomatic conditions such as hemoptysis or intractable cough occur in the absence of positive physical or roentgenographic findings, a bronchoscopic examination should be done. The portion of the tracheobronchial tree accessible to visual and "instrumentation-tactile" sense is obviously limited. When no lesion is encountered in that portion, the examiner attempts to increase his area of study by collecting smeared and aspirated

Received for publication Oct. 28, 1957.

specimens for cytologic and bacteriologic examinations. These specimens represent material coughed into the main-stem bronchi from the more peripheral bronchioles.

In the darkened room used for bronchoscopic examination it is sometimes difficult to identify adequately by marking pencils, gummed labels, etc., smear preparations collected from the right and left sides. A very simple means of identification which places the responsibility entirely upon the bronchoscopist, and has been successfully used at the Memorial Hospital is:

> *R*ight side smears are smeared at *R*ight angles to the long axis of the glass slide.
> *L*eft side smears are swabbed *L*engthwise or in the long axis of the glass slide.

This method works equally well for Papanicolaou and acid-fast smears as is demonstrated in Fig. 1. By holding the slide up to a light source, the pathologist can easily and grossly identify the side from which that particular smear was prepared.

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